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INFECTIOUS CANINE HEPATITIS (*HEPATITIS CONTAGIOSA CANIS* [RUBARTH])*

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INTRODUCTION

During the post-war years a number of major advances have been made in the field of canine pathology. As far as we in Great Britain are concerned, the findings of greatest importance have been, firstly, the demonstration of the rôle that *Leptospira canicola* plays in the aetiology of canine nephritis in this country and, secondly, the work of MacIntyre, Trevan and Montgomerie on the causes of "clinical encephalitis" in the dog other than the true distemper virus of Laidlaw and Dunkin. There is every evidence that, despite their recent recognition, these conditions have been present in this country for a long time. To this list must now be added *Hepatitis contagiosa canis* or infectious canine hepatitis, for although it is only in the last few years that it has been established as a distinct spontaneous disease of dogs, it is clear that it has existed unrecognised in this country for many years.

HISTORY

Rubarth, now Professor of Pathology at the Royal Veterinary College, Stockholm, in 1947 published the first comprehensive description of an acute, often fatal, disease of dogs, manifesting itself by distinctive and specific pathological changes in the liver, lymphoid tissue and endothelial tissues, which he named *Hepatitis contagiosa canis*. He records that his attention was drawn to the condition in 1936 by his predecessor and former teacher Professor Hjärre, who had observed the disease as early as 1928 and had described it as a "toxic liver dystrophy" of unknown aetiology. Brief accounts of the disease were given by Rubarth (1938) and Harbitz (1940). In 1945, Rubarth reported the occurrence of inclusion bodies and suggested the probable viral nature of the disease, and in 1947 he published his excellent thesis, in which he gave an extensive description of the pathology, furnished experimental proof of the infectious and contagious nature of the disease, and identified the causative agent as a filter-passing virus.

Following this work, reports of infectious canine hepatitis have been made in the United States of America by Storm and Riser (1947), Riser (1948), Coffin (1948), Chaddock and Carlson (1950) and Craige (1950); in Australia by Whittem and Blood (1949); in Great Britain by Innes (1949); and in Belgium by Florent and Leunen (1949).

The Australian and Belgian workers have succeeded, like Rubarth, in transmitting the disease to experimental puppies by the parenteral route and by contact. In 1934, Green and Shillinger showed that dogs could be infected experimentally with the virus of epizoötic fox encephalitis, and Rubarth has drawn attention to the fact that the pathological changes they described, in particular the character and localisation of the inclusion bodies, are identical with those observed in spontaneous cases of infectious canine hepatitis. The obvious close relationship between the infectious canine hepatitis virus and the fox encephalitis virus was finally established by Siedentopf and Carlson in 1949, when by serum neutralisation tests they showed the two viruses to be identical.

As early as 1930, Cowdry and Scott observed the histopathology and inclusion bodies characteristic of the disease, and the lesions described by De Monbreun in 1937 in his so-called canine distemper Type III are identical with those described by Rubarth. Evidence that the disease has been existent in this country for a number of years is given by the fact that Innes' three cases reported in 1949 on the re-examination of old autopsy material were dated 1935, 1936 and 1946 respectively. A similar search here of a collection of liver sections from canine autopsies during the period 1927-1934 yielded eight cases with a histopathology characteristic of infectious canine hepatitis. Reference to the relevant autopsy reports then showed that all these cases had clinical histories and pathological findings now suggestive of this disease, and that in no case was any other tentative diagnosis made. The recent recognition of infectious canine hepatitis by a number of workers in different countries is in full agreement with Innes' contention that a disease is never "recognised" until it has been "found" in the first place. However, it is, perhaps, striking that a disease with such a distinctive pathology should apparently pass unrecognised outside of Sweden for nearly 20 years.

INCIDENCE

Infectious canine hepatitis has now been reported in Sweden, Norway, Denmark, United States of America, Belgium, Australia, and Great Britain.

In December, 1948, the first case of infectious canine hepatitis was diagnosed here at the Royal Veterinary College, and since that time up to the end of March, 1950, 30 cases have been diagnosed out of a total of 353 canine autopsies. In a series of 151 autopsies where the dog was one year or less in age, 27 cases of the disease have been found. This is an incidence of 17·9 per cent. However, it should be pointed out that this figure does not give a true representation of the relative frequency of the disease, as this series of young dogs for various reasons included but few succumbing from distemper or "hard pad" infections. These cases of infectious canine hepatitis have originated from the following areas:—London (13), Surrey (6), Sussex (3), Devon (2), Somerset, Hampshire, Jersey, Glamorgan, Northamptonshire and Gloucestershire. When this list is added to that given by Dr. Montgomerie (1950) in his recent letter in *The Veterinary Record*, it will be seen that the disease has a widespread distribution in England.

* Paper presented to the Central Veterinary Society, May 4th, 1950.

The Clinical Picture in Great Britain

The following descriptions of the clinical syndrome, the pathology, and the epidemiological features of the disease are compiled from my series of 30 cases in an attempt to demonstrate the form that the disease takes in this country. However, it may be assumed that, unless special reference is made, the description given differs in no great detail from that given by Rubarth and other workers.

It would appear that infectious canine hepatitis can affect dogs of all ages, but, like distemper and "hard pad," it is predominantly a disease of young dogs. Rubarth showed that of 180 cases where the age of the animal was known, 149 dogs were one year or under and only 31 were two years or over. In my series of 30 cases the youngest animal was four weeks and the oldest five years. The age distribution for the series was as follows:—23 dogs were under one year; four dogs were one year old; two dogs were two years old; and one dog was five years old. The average age of the 27 dogs of one year or less was five and a half months.

The disease does not appear to possess any definite sex incidence. Seventeen of my cases were male dogs, eight were bitches, and of five animals the sex was not recorded. Of the 190 Swedish cases, 54 per cent. were in males and 46 per cent. were in females, these figures more or less coinciding with the relative distribution of the sexes in the *post-mortem* material. Rubarth has noted that in Sweden the disease appears to be commonest in the terrier type of breed; that is, in the breeds that are least susceptible to distemper, but no such breed incidence has been observed in my series of cases.

The symptoms observed in individual cases have varied considerably in nature and degree, often the only apparent character in common being the rapid lethal course of the disease. In general, the duration of illness in fatal cases has been from two to four days, although a number of dogs have been found dead in the morning having shown nothing other than apathy and inappetence on the preceding day. One animal was reported to have been playing in the garden within a few hours of its death, although autopsy revealed most extensive and marked lesions of infectious canine hepatitis. In no case with a fatal termination has the animal survived longer than seven days. It can be said that the sudden terminal collapse and death of the animal was in most cases quite unexpected even in those running a more prolonged course.

The commonest and often the first symptom observed by the owner is a pronounced apathy or lethargy, which would appear to be more profound in nature than that usually seen in the early stages of other diseases of the dog. Anorexia, or, as a rule, complete dysphagia, generally accompanies this state of depression. Abnormal thirst and vomiting, which may become bloodstained terminally, are sometimes seen. A frequent symptom, which would appear to be of considerable diagnostic importance when present, is the manifestation of abdominal pain. This may be shown by squealing or grunting in young puppies or by the curved back and the tentative manner in which the animal moves to avoid flexing its abdominal wall. Many dogs that do not evince spontaneous signs of pain will do so if the abdomen is palpated, especially if pressure is applied over the xiphoid region. It is considered that this pain is caused by the oedema of the liver and gall bladder wall that usually occurs.

During the early stages of the illness the temperature is usually raised and commonly reaches 105° to 106° F. It may remain at this level until immediately before death or the temperature may gradually drop after the first few days, becoming sub-normal terminally. It is of interest to note that temperatures of 105° have been recorded in puppies that have been in contact with affected animals although they have themselves shown no signs of malaise at any time.

Diarrhoea is often observed, although several cases have shown complete bowel stasis during the illness. The presence of blood in the vomit or faeces is considered to be a grave sign and generally indicative of impending death.

Both Rubarth and Coffin have commented on the occurrence of enlarged and reddened tonsils, which often suggests a clinical diagnosis of acute tonsillitis. While enlarged and often haemorrhagic tonsils were described clinically only six times in this series, they were found in the majority of cases autopsied here. Similarly, although an enlargement of the sub-maxillary lymph nodes was only reported once clinically, we found it *post-mortem* in almost every case, the degree of enlargement often being extremely marked.

The mucous membranes were usually pale and anaemic in appearance, especially when the disease had assumed its haemorrhagic character. Occasionally, petechial haemorrhages were to be found on the buccal mucosa. Despite the extreme degree of liver damage present in most cases, jaundice was seen in only five out of the 30 animals. A slight cough, dyspnoea and tachycardia were sometimes present as a result of cardiac insufficiency and lung oedema. The subcutaneous oedema of the head and neck described by Rubarth was observed in two cases only at autopsy. Lachrymation was observed in four cases, and one animal had a unilateral central opacity of the cornea. This lesion has been observed by Rubarth in three non-fatal cases of infectious canine hepatitis and also by Green *et al.* (1930) in non-fatal cases of epizoötic encephalitis in foxes.

Clinical evidence of involvement of the central nervous system was apparent in four cases that showed incoordination of the hind limbs and epileptiform fits and in one case that had a fit immediately before death. No non-fatal cases were reported to have shown nervous symptoms.

Morbid Anatomy

In the majority of cases the autopsy findings were sufficiently characteristic to enable a tentative diagnosis of infectious canine hepatitis to be made, although a definite diagnosis was given only on the histological demonstration of the specific inclusion bodies in the liver and other tissues. The general *post-mortem* picture can be said to vary according to whether the disease has assumed its haemorrhagic character or not. Thus many cases, particularly young puppies, apparently die before bleeding commences and at autopsy the morbid anatomical picture, apart from the liver lesion, is essentially one of circulatory failure and oedema, whereas in other cases this picture is obscured or overshadowed by the widespread and extensive haemorrhagic lesions present in the various tissues. The following lesions were commonly observed *post-mortem*:—

(1) The mucous membranes were generally pallid, especially when haemorrhages were present in the body. Jaundice was rare and observed in only five cases.

(2) Subcutaneous oedema of the neck and sternal region was seen in three cases, and large subcutaneous haemorrhages were present in a further three animals.

(3) The serous cavities usually contained a varying quantity of fluid. That in the peritoneal cavity was usually bloodstained, approximating to whole blood in a few cases.

(4) Usually the myocardium was pale and flabby and the right heart showed some dilatation. Extensive subendocardial haemorrhages were found frequently in the left ventricle, and the atrioventricular valves, particularly the tricuspid, were often oedematous. In three cases subepicardial haemorrhages were present.

(5) The lungs as a rule were un-collapsed and extremely oedematous, the trachea and bronchi then being filled with frothy fluid.

(6) Although the condition is always one of hepatitis, the gross appearance of the liver has varied considerably from case to case. Thus in three animals no macroscopic liver changes were observed, while another showed a massive liver necrosis of the kind described in the human as acute yellow atrophy. In general, however, the liver was paler than normal and possessed a prominent lobular pattern. Sometimes it was enlarged and swollen. On occasion, where central lobular haemorrhage was marked, the liver resembled the " nutmeg " appearance of chronic passive venous congestion. It was frequently observed that a cut surface remained dry and matt despite the obviously high blood content of the liver. The capsular surface of some livers showed a fibrinous deposit of varying thickness, which was most marked between contiguous lobes. In some 50 per cent. of cases the wall of the gall bladder was grossly thickened by a subserous oedema, sometimes approaching $\frac{1}{2}$ in. in thickness.

(7) The spleen was usually enlarged, dark red in colour, and slightly diffluent.

(8) Most young dogs showed a marked oedema of the thymus, often accompanied by petechial haemorrhages. Many older dogs in which the thymus tissue had almost disappeared still showed a marked oedema in this region of the anterior mediastinum.

(9) The stomach and small intestine sometimes showed subserosal and submucosal haemorrhages. Often the contents were bloodstained, and in a few cases the lumen of the intestinal tract was filled with apparently pure blood, even when no macroscopic haemorrhages were to be observed in the stomach or intestinal mucosa. The wall of the gut was generally thickened by oedema, and one puppy showed an extensive agonal intussusception.

(10) In all the cases autopsied the lymph nodes have been enlarged and oedematous, and usually the supra-sternal, portal and mesenteric nodes have shown some signs of haemorrhage into the medulla. In many cases all the visceral and carcase lymph nodes have been grossly enlarged and saturated with blood.

(11) Associated with these changes in the lymph nodes, the tonsils have usually been enlarged and oedematous and protruding from their crypts. Sometimes they were congested and at other times frankly haemorrhagic.

(12) The meningeal vessels of the brain were generally hyperaemic and in six cases extensive subarachnoid haemorrhages were present over the base of the midbrain and cerebral hemispheres.

Histopathology

Liver.—Histologically the liver lesions have varied from merely mild regressive changes to a massive necrosis resembling acute yellow atrophy in one case. However, in general, the liver has shown marked dilatation of the sinusoids, often with haemorrhage, in the central lobular zone. The hepatic cells were usually partly or completely necrotic in the central lobular region, while the peripheral cells of the lobule were either intact or showed only mild degenerative changes, usually manifested by karyorrhexis or by " margination " of the chromatin of the nucleus and a vacuolated appearance of the cytoplasm. The central cells undergoing necrosis appeared dissociated and rounded-off, showed a granular dissolution of the cytoplasm, and tended to have a marked staining affinity for eosin. (Fig. 1.) The sinusoidal endothelial cells and Kupffer cells were usually swollen and very prominent.

Large, usually basophilic, intranuclear inclusion bodies were found in hepatic epithelial cells, in the endothelial cells of the sinusoids, and in the Kupffer cells. (Fig. 2.)

Tonsils, lymph nodes and spleen.—These have commonly shown hyperplasia of the non-lymphoid elements, oedema or haemorrhage in the sinuses, and oedema and necrobiotic changes in the lymphoid follicles. Intranuclear inclusion bodies resembling those found in the liver were seen in reticulum cells, sinusoidal littoral cells, and in the vascular endothelium of the sheathed arteries of the spleen.

Kidney.—Inclusion bodies have been found in the capillary endothelium of the renal glomeruli, while merely mild degenerative changes were to be seen in the kidney parenchyma.

Brain.—The blood vessels of the brain and meninges often showed a marked swelling of the endothelial cells with accompanying necrobiotic nuclear changes. Inclusion bodies have been found in these swollen vascular endothelial cells, but never in any type of nervous tissue cell. No true encephalitis or evidence of damage to the nervous tissue elements, other than that caused by the vascular damage such as oedema and subpial haemorrhage, was ever observed.

Inclusion bodies.—Those found in infectious canine hepatitis are unique in so far as they occur in tissue derived from primitive entoderm (the hepatic parenchyma) and from primitive mesoderm (the vascular endothelium, reticulum and sinusoidal cells).

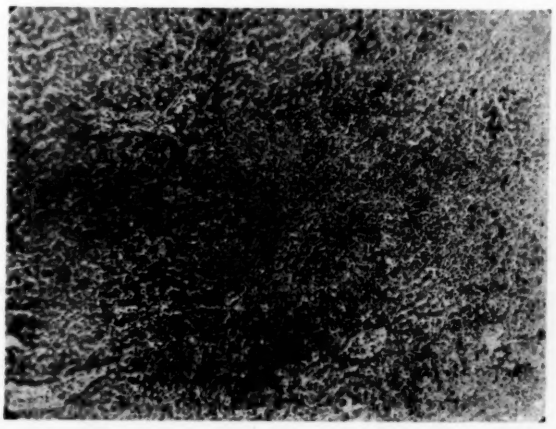


Fig. 1.—Infectious Canine Hepatitis. Liver (Low Power) H. and E. An area of central lobular necrosis showing dissociation of liver cells and hyperaemia and haemorrhage.

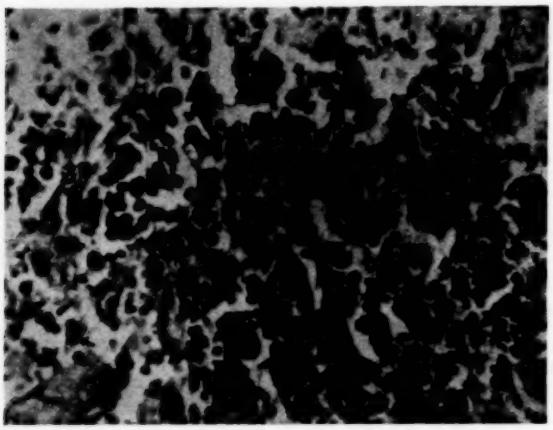
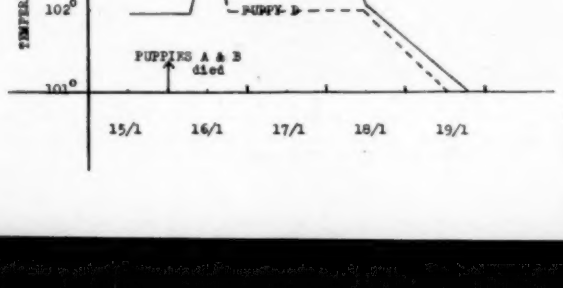


Fig. 2.—Infectious Canine Hepatitis. Liver (High Power) H. and E. Lacunose dilatation of the sinusoids with dissociation and atrophy of liver cells. The majority of liver cells are showing margination of the chromatin on the nuclear membrane and contain large intranuclear inclusions (arrowed).

Epidemiology

Infectious canine hepatitis presents several unique factors when compared with other virus diseases for, despite the fact that in certain cases it may pursue an extremely rapid lethal course, it has become apparent that the mortality rate is actually quite low. This striking fact is exemplified in most outbreaks. Thus a typical history is for one or two animals of a litter to sicken suddenly and die, a few other litter mates may show symptoms for two or three days and recover, and the remainder of the litter together with other in-contact puppies exhibit no signs of ill-health at any time. In 15 of the outbreaks in my series the disease occurred in kennels or on premises where more than one dog was kept. In 13 of these, other dogs showed signs of illness, but in only eight cases did more than one animal die. An illustration of the unusual sporadic nature of the disease is, perhaps, best given by a study of a typical outbreak in breeding kennels containing some 30 dogs. A litter of four Labrador puppies, aged six weeks, were weaned on the evening of January 14th, 1950. At 8 a.m. the following morning one puppy was squealing and showing signs of abdominal pain and refused its food. The other puppies appeared healthy and ate normally. At 11 a.m. a second puppy developed symptoms and both puppies died that night. The temperature charts of the two remaining puppies C and D are shown.



At the time when its temperature was 105° puppy D showed no clinically observable symptoms, but later both puppies C and D developed slight coughs and sore throats. By January 20th both puppies were completely recovered. During this time a litter of Springer Spaniels of the same age were in an adjacent kennel with a connecting door, and the kennel maid always cleaned this kennel after cleaning that of the Labradors, using the same tools. These puppies showed no signs of illness at any time, nor did any other animals, despite the fact that the mother of the infected puppies exercised with all the other dogs.

It would appear from the character of the naturally occurring disease that there is a marked familial natural immunity to infectious canine hepatitis. This theory is supported by the fact that Rubarth found it impossible to cause symptoms of illness, other than perhaps a mild pyrexia, in any of the puppies of certain of his breeding bitches. The sera of these puppies were negative for immune bodies before infection, but were uniformly positive afterwards. Green, similarly, found that whole litters of puppies were either highly susceptible or highly resistant to infections with the virus of "fox encephalitis."

The development of immune bodies in animals after exposure to the virus, even in those possessing an apparent natural immunity, provides a means of assessing the frequency of occurrence of the disease. Using the complement-fixation test, Rubarth found that in Sweden 70 per cent. of dogs of varying ages possessed antibodies to infectious canine hepatitis. In Belgium, Florent and Leunen obtained a comparable figure of 60 per cent.; they failed to demonstrate complement fixing bodies in the sera of puppies under two to three months of age. It is clear from these figures that the disease is of widespread occurrence in Sweden and Belgium, and it is not unreasonable to suppose that a similar state of affairs exists in Great Britain. Appreciation of these important findings helps towards an understanding of the apparent peculiarly selective nature of the disease. However, while it is not difficult to envisage the occurrence of an hereditary natural immunity in some families, it is not easy to account for the frequent marked variation in sensitivity to the disease exhibited by puppies of the same litter.

Diagnosis

All the cases in this series have been diagnosed on the histological demonstration of the specific intranuclear inclusion bodies in the parenchymatous cells and sinusoidal endothelial cells of the liver. No transmission experiments or serological investigations have been made.

Rubarth always succeeded in demonstrating the presence of antigen in the liver in positive cases by means of the complement-fixation test. He also was able to demonstrate antigen in the sera of a few cases during the febrile stage of the disease. Florent and Leunen, similarly, were able to detect antigen in the sera of a few of their experimentally infected animals and they found antigen in the urine of one case. However, owing to the rapidly lethal course of fatal cases and to the practical difficulties of carrying out and interpreting the complement-fixation test, it is difficult to see that this test will be of much value as a means of clinical diagnosis.

A finding of some therapeutic and, indeed, diagnostic value is that of Craige (1950), who has recently reported that the profound depression and coma observed in dogs suffering from acute infectious canine hepatitis is probably

due to a hypoglycaemia resulting from the liver insufficiency. Such dogs make a spectacular, but, unfortunately, only temporary, response to the intravenous injection of glucose. However, he reports that if this is then followed with injections of hyperimmune fox encephalitis serum the majority of cases make immediate and uneventful recoveries.

Summary

A review of the literature of infectious canine hepatitis has been given and clinical and pathological data compiled from 30 cases of the disease diagnosed at autopsy at the Royal Veterinary College between December, 1948, and March, 1949, have been reported.

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Discussion

Miss Joshua (the President) said that they appreciated Dr. Montgomerie's action in postponing the preliminary publication which he and his colleagues had proposed to make in the veterinary Press in order to give them the benefit of their observations at that day's meeting.

Dr. R. F. Montgomerie paid tribute to the work of Mr. Pay and said that so far as he and his colleagues were concerned they were pleased to occupy the secondary rôle of opening the discussion. It should be realised that Green in America was the pioneer and it was really he who established the existence of this disease, although he called it fox encephalitis. He did show the transmission of the disease to the dog and he laid a sound basis for those who followed in recent years. Rubarth's monograph was quite a classic and one was impressed by his approach to the problem. What were they going to call this disease? They could not take priority grounds and accept "fox encephalitis" as they now knew that the disease occurred in animals other than the fox. There was a suggestion that they should accept Rubarth's name for it, "*Hepatitis contagiosa canis.*" He thought it would be a little unfortunate if they called it "Rubarth's disease," because if one wanted to attach any worker's name to the disease they would have to go back and call it Green's disease. He liked "*Hepatitis contagiosa canis*" but thought Mr. Pay's preference was "infectious canine hepatitis." It might be interesting to have some suggestion out of the discussion as to what they were really going to call it. The "Central" might well take the responsibility rather than workers who were apt to make mistakes in naming diseases of the dog!

He represented a team of workers and he wanted it appreciated that his remarks represented their work and their findings. At the Wellcome Laboratories they had been aware of Green's work since 1930 and had kept a look-out for the occurrence of this disease in Britain. They had not seen a case until March, 1947. That was in a foxhound puppy with the characteristic history of collapse, sub-normal temperature and death within six hours. The *post-mortem* appearances were characterised by little more than an enlargement of the spleen, congestion of all the organs and particularly the lymphatic glands. The pads were soft and tissues injected into ferrets caused no reaction. The histological picture was clearly that of *H.c.c.* Although they received a large number of carcases, there was no other instance of this disease until a year later. At that time they thought the disease of rare occurrence and only mildly contagious to the dog. It was recorded as a pathological curiosity. To-day the evidence was quite different and indicated the widespread presence of the disease in this country. One had some reason to believe that many dogs were affected; some showed little clinical effects and many did recover. The second occasion on which they diagnosed the disease was in a kennel of Scottish terriers where the dogs were kept under conditions of scrupulous cleanliness and where there was a great deal of washing down of the kennels with carbolic disinfectant. The first death was ascribed to carbolic acid poisoning. Later, the alternative diagnosis was hard pad disease. A number of deaths occurred. An interesting point was that collapse was quite a prominent symptom; death occurred in six to ten hours. When tissue from dogs in that outbreak was injected into ferrets the latter went down to hard pad disease in 19 to 31 days. This seemed of some significance, but it was later realised that hard pad disease had appeared as a natural disease among British ferrets. Since that time they had had many cases of *H.c.c.* and believed there had been a wave of infection with the virus. They were prepared to accept that the disease had been present in Britain for some years but contended that it was of low incidence until late in 1948. It was possible that many veterinary surgeons in the country had in recent years missed the true diagnosis of the *H.c.c.*, ascribing the illness to nervous forms of distemper, so-called hard pad disease or to poisoning in cases where there were early deaths and no evident symptoms. The difficulties which veterinary surgeons faced were well illustrated in one of the first outbreaks they encountered. The first case showed fits almost continuously, temperature 103°, tonsils enlarged and some enlargement of the lymphatic glands. The treatment was little more than sedative, yet it recovered. The second puppy had continuous violent convulsions and was destroyed. The remaining three puppies of the litter showed tonsillitis and a marked temperature reaction of 104° or a little more. Those three puppies were given 30 c.c. of hard pad serum intravenously. They recovered. That this was an outbreak of *H.c.c.* was based on the fact that the carcase of the puppy which was destroyed had the typical *post-mortem* appearances of *H.c.c.* He had referred earlier to the findings in their second case, where the ferrets inoculated with tissue of the dog with *H.c.c.* had gone down with hard pad disease. That had happened on a number of occasions and had led to a high level of confusion. They were now conscious of the fact that the ferret population of this country during the last five or six years had become fairly extensively infected with what they called the virus of hard pad disease. Previously, dealers' ferrets need only be held in isolation for two weeks; by the end of that time they would be dead from distemper or could be accepted as healthy ferrets suitable for their work. During the course of the last few years they had repeatedly had the experience of purchasing ferrets, keeping them in good isolation, yet having them go down with hard pad disease in three weeks or even longer; the record was 40 days. They had reported finding hard pad disease in carcases which had reached the laboratory and now knew that some of the ferrets they injected with the emulsion from the tissue of dogs were already carriers of the hard pad virus. They came to the conclusion that, where they were using the ferret to prove the presence or otherwise of a virus disease in the dog, the ferrets must have been specially bred and reared in circumstances which excluded the possibility of extraneous infection. Laidlaw and Dunkin had met the same difficulty in dogs during their classic work on canine distemper. He (Dr. Montgomerie) and his colleagues had occasionally produced a condition which was accepted as hard pad disease in ferrets inoculated from the tissue of dogs which died of *H.c.c.* but they did not accept that as a true bill. It was possible that the injection had stirred up virus the ferret was carrying or that the virus had become active during the period of observation. Possibly a similar state of affairs existed in the dog population; there had been occasions when the injection of material which could not have contained any virus had produced a classical case of hard pad disease in the dog. It seemed that some of the dogs were carriers of that virus and the injection of something extraneous lit up the infection and produced the clinical condition. It was possible that some dogs were carriers of the virus of *H.c.c* and that an injection might stir up the virus and produce clinical disease; a dog injected with distemper vaccine might die of *H.c.c.* within a short period of that injection. Green regarded this disease in the fox and the dog as similar but noted a tendency for the appearance of the discharge from the eyes and nose to become purulent in dog cases of *H.c.c.* They had not

seen sufficient cases to say whether they agreed with that observation, but if that was so the difficulty of clinically differentiating between *H.c.c.* and distemper was even greater. He hoped Mr. Pay would deal with the natural spread of the disease. The experience of other workers with *H.c.c.* did not appear to classify it as highly infectious but it might be a little dangerous to come to that conclusion. If it were true that some infected dogs showed little clinical evidence, it was difficult to tell how infectious the disease was or the manner in which it spread. The reason why many practitioners had not had experience of the disease might be because of low infectivity or a tendency of the virus to produce an immunity without obvious clinical disease. It was only by a serological survey of the dog population in this country as was done in Sweden and Belgium that they would get good knowledge of the extent of the disease. It was only by the use of suitable tests that they would be able to watch the spread of the disease in this country and in various kennels. Until that was done it was unsafe to dogmatise on features relating to the spread of *H.c.c.* Green had suggested that infection could hang about premises for three years. Experimental transmission was difficult. They had not yet transmitted *H.c.c.* in its full clinical form. Green found difficulty in transmitting the disease and Rubarth produced good evidence to ascribe failure to the fact that some of the puppies he used had a high level of immunity. Their own failure arose from a similar reason; some little time ago they discovered that the kennels from which they obtained the test puppies—kennels as completely closed as they could be—undoubtedly had *H.c.c.* present in them. They had seen typical cases from certain litters and they had good evidence that a sub-clinical form of the disease was fairly extensive among puppies from those kennels. Until they developed tests for determining whether the animal was resistant or otherwise they could not expect any real level of success. The question would be asked, "What have we to control this disease?" Until they were able to establish methods of transmitting the disease, methods of active immunisation could not be worked out. Green had used a mixture of virus and serum injected into dogs as a means of identifying the virus. In a number of cases the dogs did not go down with the disease until some time after the injection. It seemed that the virus was able to remain active in the body until such time as the serum was eliminated or some extraneous circumstances stimulated the virus to activity. A virus-serum method seemed out of count. One had seen *H.c.c.* confined to certain litters and one was tempted to suggest that it might be possible to immunise the bitch and so protect the young puppies, for there was evidence that puppies from some bitches were quite resistant. It was remarkable that in some outbreaks of *H.c.c.* only one or two deaths occurred in certain litters; a few other puppies showed a rise in temperature and nothing further. That was the experience after watching the position carefully in kennels where the disease had expressed itself by a high death-rate in certain litters. When one was speaking of a disease, yet very much in the dark regarding certain features, it was difficult to make a material contribution to the discussion. The pathological picture seemed to be fairly clear but the epidemiology was in the wilderness and the question of protection was something for the future when they had passed through that wilderness.

Miss Joshua said that this disease was not merely a pathologist's dream—it was a clinician's entity and it was going to be a vital matter in canine practice. They should not hesitate to put their experiences, even if small, before the meeting.

Mr. C. G. Hopson wondered whether recognition of the existence of this disease in this country accounted for some of the sudden deaths he had had in his practice in the last few years. He remembered two cases particularly well in 1946, one a mongrel sheepdog aged four months, the other a three-and-a-half-month-old Scottie. Both were taken suddenly ill with hign temperature (106°) and great apathy. As Laidlaw-Dunkin was the only virus disease recognised at that time, both received distemper serum (10 c.c.), but died within 36 hours.

More recently (February of this year) a three-and-a-half-month-old Cocker spaniel died within 24 hours of first being seen. Here the symptoms had not been so marked: occasional vomiting and diarrhoea, temperature 103·5°, but again very great depression. *Post-mortem* findings showed a grossly enlarged liver—mottled—with fibrinous shreds on and between the lobes, gall bladder enlarged and pale in colour, mesenteric lymph glands enlarged and sanguinous. Portions of the liver and lymph glands were sent to Dr. Montgomerie, who had reported back that it was a case of *H.c.c.*

It was of interest to note that the puppy had been wormed shortly before the onset of symptoms with a mixture containing ol. chenopodium; Rubarth, in his monograph, had noted that worming with tetrachlorethylene had preceded the onset of symptoms on some occasions, and it seemed possible that the use of any ascaricide had acted as a trigger mechanism in latent infections. None of the subacute cases mentioned by Craige had been seen.

Mr. Hodgman: They had recently had an epidemic of *H.c.c.* at the breeding kennels of the Canine Research Station at Newmarket and the general symptoms differed little from the description given by Mr. Pay. So far they had failed to transmit the disease under laboratory conditions. Mr. Parry had written a paper on the outbreak, which he believed would be published soon, and it would be interesting to compare his findings with those of Mr. Pay. (See following paper in this issue.—*Editor.*)

Dr. Edwards: Virus research, upon which he had spent a great deal of his time, was, depending upon whether the outlook of the worker was one of mere exploitation or one of genuine exploration, either extremely simple or was tedious. It was with pleasure that they had heard Mr. Pay, now a young worker just entering the field, deal with the subject in the latter respect, with confidence and with assurance. He (the speaker) sincerely hoped he would continue on a line of research which was almost certain to bear fruit in the future. They had expected good and mature critical comment from Dr. Montgomerie and they had not been disappointed. To both these able speakers they were now very grateful. What was the disease which now seemed to have become familiar in literature as "Rubarth's disease"? He had followed Green's work in America closely and critically. What was certain was that it was he who had identified it, and experimentally only, in dogs as a peculiar disease arising from artificial infection with fox encephalitis, a well-known natural disease of foxes. But they had to be grateful to Rubarth for recognising it as a natural disease of dogs and putting on record such an excellent comprehensive description of it, with its peculiar train of symptoms and curious natural history. It was something new, they were consequently made to realise, and different from distemper, and it complicated the clinical distemper picture as they had previously thought it to be. The simple problem of distemper, as they thought it was in England after the Laidlaw and Dunkin research, was no longer a matter of simplicity, for they had many diseases, like Rubarth's disease, Pugh's brown mouth or dry distemper, and now probably other virus diseases as well, complicating the field picture immeasurably so far as the clinical categories of canine disease were concerned. The whole subject of canine diseases into one category of which fell true virus distemper ("Carré's disease" as the French, with good reason, called it) needed reopening and reinvestigating. Had they anything comparable in the virus world in other species which could throw some light comparatively on this extraordinary condition of Rubarth's disease, a condition which did not seem to be easily transmissible and which produced visible symptoms in a few animals only in a group, but which, when they were observed, proved that they were alarming? Recently in his comparative studies upon rinderpest, or cattle plague, he was struck by the excellence of Green's work in America, to which Dr. Montgomerie had paid tribute. There was no question but that canine distemper as a virus infection seemed to be the twin brother in the canine species to rinderpest in the bovine species. Had they in the bovine anything similar to Rubarth's disease which might reveal something of the nature of that disease? In cattle it had just occurred to him that they had it in malignant catarrh, a virus disease which was only transmissible experimentally from the affected to the healthy with difficulty. In fact, experimentally, so rare were cases of actual transmission that one had to go to great pains to explain how the disease arose at all. In recent years some workers had shown that a mild, usually almost symptomless, disease might prevail among sheep in localities in which malignant catarrh broke out sporadically in cattle and it was the transmissible mild sheep infection which produced in cattle the severe, but non-transmissible, disease. They had other, similar, instances of mild infections which, when transmitted to an aberrant host, set up severe disease. Such instances included the so-called "swineherd's disease" and several tick-borne rickettsioses. Might it not be that the reservoir of Rubarth's disease was the fox, and that when the dog contracted the disease it did so sometimes in a severe and malignant form as the bovine did when it contracted malignant catarrh. Furthermore, the chances of severe clinical attack might depend, as Mr. Pay had suggested, upon a familial susceptibility to a condition of grave hypoglycaemia consequent upon liver damage caused by such adventitious infection of the dog with virus from an extraneous reservoir. He threw that out as a speculation. At this stage they were only at the beginning of a reopening and reinvestigation of the canine virus diseases but it was a field which would engage young men like Mr. Pay and Mr. Henderson for many years to come.

Mr. Platt said he was interested in the two papers and he would like to congratulate Mr. Pay on the excellent account of the pathology he had given; it was surprising that a disease with such characteristic features should have escaped notice for so

long. He was interested in Dr. Montgomerie's remarks about the infection of the stock of ferrets with hard pad and he would like to ask on what criteria that diagnosis was made. Also whether the condition was readily transmissible to dogs. One was impressed by the extreme complexity of the problem of distemper and hard pad and their relation to each other, on the one hand, and the exact significance of *H.c.c.* virus and Green's fox encephalitis virus in the dog on the other. From the American work, it would certainly seem that the two latter viruses were very closely related.

Mr. True asked whether the distemper serum put up to Green's specification in America had had any effect on the infectious canine hepatitis where it had been tried. He explained, in answer to Dr. Montgomerie, that he meant Green's distemper serum and not distemper virus.

Mr. W. A. Pool spoke about the general characters of *Hepatitis contagiosa canis* as described in the literature and as described by Mr. Pay and Dr. Montgomerie. The fact that when the disease occurred there appeared probably to be a high morbidity and a low mortality and that death tended to occur without an appreciable period of illness gave a picture that was different from that of most virus diseases. In certain virus diseases complicating factors sometimes provided a situation where the disease caused by the pure virus was benign, while the natural disease in which such complicating factors operated naturally produced a very serious illness. Louping-ill in sheep and swine influenza were examples. Was *Hepatitis contagiosa canis* an example of such complicating causal factors?

The recent observations in poliomyelitis in human beings that the incidence of clinical illness accompanied by paralysis of an arm had been shown to be related to an injection into the infected arm, a few days before the development of the paralysis, of a vaccine against diphtheria or whooping cough was of great interest. Here was an easily understood factor that had precipitated a virus infection. In this field of pathology evidence was accumulating that helped to explain the natural history of some diseases. A lot more information was required about this dog disease and one line of enquiry that appeared to be worth study was the whole range of circumstances related to its occurrence.

For the production of irrefutable evidence concerning the nature of the disease it would appear that, whatever laboratory animals might be found suitable for work involving its transmission from animal to animal, experiments would require to be carried out finally on dogs. For such work a large supply of dogs was necessary. The dogs would have to be kept in such circumstances that they were devoid of any of the effects of previous experience of contact with the disease. For this the very elaborate system used in the dog distemper enquiry was necessary. Speakers had referred to great variations in the susceptibility of dogs that had been used in experiments on *Hepatitis contagiosa canis*. It might be that there were susceptible and resistant breeds or strains of dogs. To provide an adequate supply of the right type of dog at the place required, at the time required and in the number required would be a very great task, but for this and some other disease problems it would have to be faced. This was now an everyday procedure in some laboratories with regard to smaller laboratory animals and the history of the work done had shown that while it could be achieved there were often great difficulties to be surmounted

The Replies

Mr. Pay: It was extremely interesting to hear Mr. Pool's remarks concerning the possibility of infectious canine hepatitis being analogous in some respects to virus infections such as louping-ill or swine influenza, where some "secondary factor" played a part in the pathogenesis of the disease. When one studied the peculiar epidemiological features of infectious canine hepatitis it made one strongly suspicious that some precipitating factor or factors existed which could bring about a clinical infection, although apparently it could be fully reproduced with pure virus and no co-existing rickettsial or bacterial infection had been demonstrated. Without such a suspicion it was difficult to understand how the virus could give rise to such a rapidly fatal disease in some animals and yet merely stimulate the production of immune bodies in some 60 to 70 per cent. of dogs. Hereditary factors, it seemed, played an important role in the susceptibility or resistance of the progeny of certain bitches, but it was not yet clear whether this was in fact due to a true hereditary natural immunity or to an acquired passive immunity obtained *via* the colostrum. Assuming for the time being that such an immunity existed, it was relatively easy to understand how the virus could affect the puppies of one litter and yet give rise to no observable symptoms in other in-contact puppies. However, it was difficult to account for the marked variation in susceptibility to the disease that was frequently encountered among animals of the same litter. One would have thought that all factors such as the serological or nutritional status of individuals in a litter of suckling puppies were more or less equal, yet it remained clear that something caused some puppies to die, some to recover after showing more or less typical symptoms, and others to show no apparent signs of ill-health. The nature of any possible precipitating factors would probably remain obscure until carefully controlled experiments could be carried out on susceptible dogs bred under the conditions that Mr. Pool had outlined.

It was probably a suitable place to mention that Rubarth had commented on the fact that several of his naturally occurring cases had followed the administration of anthelmintics of the tetrachlorethylene type. Two of the cases he (Mr. Pay) had found by examining old autopsy material at the Royal Veterinary College had occurred after the use of oil of chenopodium. What was the story in these cases? Did the liver damage brought about by these drugs act as a "trigger mechanism" on a present, but latent, virus, or did it merely pave the way for a coincidental infection?

Dr. Montgomerie, who also replied, dealing with the suggestion that the disease had gone unnoticed, said that he doubted it. He felt confident in saying that it had not, as this disease at the moment was primarily recognised on pathological features which were quite distinct; he was not a pathologist, but he would be very surprised if it had been missed. He was not prepared to accept that the disease had gone unnoticed as far as the material reaching the Wellcome Laboratories was concerned. They saw cases in 1947 and 1948 and since then a large number. It had occurred in 39 cases as previously recorded. He thought that the infection had been present in the country, but only of low incidence until the recent wave appeared. The disease he had mentioned in the stock ferrets was diagnosed on symptoms and on transmission to the dog, but not as a clinical disease. It was different from distemper. In the pre-war days they used to hold ferrets in isolation for 10 to 14 days, and then some were put in the infected ferret house as controls. On many occasions the same pair of ferrets acted as controls for a long period, did not become infected and were later proved susceptible to distemper. To-day they put ferrets in isolation and held them there, yet some did go down with hard pad disease after a long incubation period. Green's fox encephalitis, Rubarth's *Hepatitis contagiosa canis* and the disease Mr. Pay had described were one and the same thing. They based their belief on pathological evidence which was good. Mr. Pool had suggested that there might be some other virus or organism complicating the picture. It was remarkable that in some cases *H.c.c.* virus caused a mild sub-clinical disease only and in other cases a disease so violent that the dog might be dead within 12 hours. That was not so very uncharacteristic of some virus diseases. At the moment he did not quite see the need to postulate some other factors, but he allowed the possibility that nutritional conditions were associated with these great differences. There was good evidence from the human field that injection of a product, such as diphtheria prophylactic, precipitated occurrences; not always general but sometimes local. He thought this illustrated fairly well what might happen in virus diseases of the dog; virus was present in the body and was activated by some such action as an injection of vaccine. Mr. Pool had suggested breeding susceptible animals, but the difficulty was to produce animals that had not got some virus infection before they started using them. That was the outstanding difficulty about the whole thing. It was easy to talk about basic principles, that one must get animals free from virus diseases, but few people kept kennels of dogs without having accidental infection in them. What they were trying to do was produce a really large breeding establishment of ferrets free from virus disease. Their ferret colony produced about 3,500 a year, about half the number they wanted for their work.

Further Discussion

In further discussion, Mr. Hodgman said that they were in point of fact trying to do exactly what had been suggested, *i.e.*, breed dogs in isolated kennels, free from any immunity. Whether they would be successful or not remained to be seen, but it was no mean problem. To do this meant having complete isolation of the dogs, with particular attention to food from a sterilisation point of view, and, in addition, very strict control over the personnel working with these dogs. They had a colony of 50 or 60 dogs and twice already they had epidemics amongst them which necessitated their starting all over again. He thought that *H.c.c.* had been seen by G.P.s for some time but that its significance had not been recognised. Not that there was any connection, but it struck him that there was close similarity between some of the symptoms of *H.c.c.* and the so-called infective gastro-enteritis of greyhounds. There was the picture of the greyhound racing and being dead in the morning and the *post-mortem* showing a condition which

could not possibly have occurred in so short a time. Having seen on *post-mortem* the enteritis present, it appeared impossible that that dog could have raced the night before and sometimes even won.

Dr. Edwards said that it was the first time that this important disease had been discussed in this country. It reminded him of the investigations regarding Johne's disease and the occasion in 1904 when Bang was invited over to address the N.V.M.A. annual meeting in Liverpool and there described a new disease. It was reported and immediately practitioners were on the look-out for this disease, and before many years a wealth of experience had accumulated to show that the disease was actually widely prevalent in this country and was caused by a bacillus that was different from that of tuberculosis. It was probable that the virus of Rubarth's disease belonged to the same group as that of true distemper, but it was now almost certain that as soon as practitioners were made aware of its characteristics they would get the disease well placed within a few years. Mr. Pay's paper had been presented in such a comprehensive and readily intelligible way that readers of *The Veterinary Record* would be on the look-out for this new and hitherto unfamiliar condition. The great experience of Dr. Montgomerie had bolstered Mr. Pay up further and that would put them right in regard to the aetiology and the complexity of the problem. From the point of view of canine practitioners it was, he thought, an historic occasion, as much historical as that famous address in Liverpool in 1904 in regard to Johne's disease, and as a result, he thought, the whole of the profession would be on the alert to discover what might be as important in the canine world as Johne's disease proved to be in the bovine world. He proposed that they close the discussion at that stage; that would give them proper time to contemplate carefully the evidence after it had been published and then they could come back again after they had been able to correlate it with their experiences garnered in practice.

Miss Joshua said that it had been suggested that they should try to find some name for the disease, which would be widely acceptable.

Miss Uvarov asked whether Mr. Pay could tell them more about the sub-clinical cases. From the discussion it sounded almost as if it was the clinicians that might miss these cases, and if Mr. Pay could tell them more what they would have to do in all sub-clinical cases they would be much happier. Mr. Hodgman had suggested that this condition was similar to infective gastro-enteritis in greyhounds and that they should bear it in mind. Dr. Edwards was right in suggesting that they should go carefully.

Mr. Hodgman said he only drew attention to the similarity of some of the findings and rapid death. He was not now suggesting that *H.c.c.* and infective gastro-enteritis was one and the same thing.

Miss Uvarov remarked that infective gastro-enteritis was alleged to be due to so many things and this was yet one more alleged cause of it.

Mr. Pay said that his knowledge of the symptoms shown by in-contact puppies had been limited to the information supplied by practitioners when submitting cases for autopsy. There was, of course, no proof that these other puppies had been infected with infectious canine hepatitis other than the fact that they had been in contact with a fatal case. However, from the clinical histories he had examined it appeared that such puppies could respond to infection in a variety of ways. Thus, some had been observed to have had temperatures of 105° or 106° although they showed no signs of malaise, others had shown mild symptoms of abdominal pain and tonsillitis, and others had exhibited symptoms almost equal in severity to those seen in fatal cases.

Mr. McLauchlan said that the sort of dog with ten days diarrhoea, temperature 105° and vomiting with blood was seen in London clinics every day.

Dr. Montgomerie assured Miss Uvarov that his use of the term sub-clinical was not a challenge to his practitioner friends, for he believed this disease did occur in a form in which the only "symptom" was some rise in temperature. As far as they were concerned the diagnosis at present was based on *post-mortem* evidence. He was not going to make a prophecy, but they hoped to find a diagnostic test; possibly it might only indicate that the dog had had *H.c.c.* That was not very helpful to the practitioner, but they were at that stage when it appeared probable that many dogs became infected with the virus, showed no illness, but became immune. They were satisfied that they had had this disease among puppies under careful observation for a time before deaths and *post-mortem* evidence made it possible to recognise it. Since then there had been other cases, but the interesting fact was that when survivors which had shown no symptoms reached the laboratory they were found immune. He would say the practitioners were missing something, but something they could not determine by clinical means.

Mr. Evans: In going over the whole ground, was he right in supposing that this disease was more likely to occur in a group of animals in kennels or more in isolated cases. He had had two cases which he would have missed had it not been for the insurance companies' request for a *post-mortem* examination, and he had had to send the carcase to Mr. Pay and he put him right. Both those cases were completely isolated and the dogs seldom allowed to mix with other dogs.

Mr. Pay said that 15 of his cases had occurred on premises where only one dog was kept and the other 15 cases had occurred in kennels. Thus it appeared that the single dog was as prone to infection as the dog in kennels.

With regard to diagnosis at autopsy, a rapid diagnosis could be given in the majority of cases where hepatic intranuclear inclusions were plentiful by the examination of Giemsa-stained smears of liver tissue. With experience, practitioners should find no great difficulty in differentiating true inclusion bodies from the hepatic nucleoli which were quite large in smears.

Col. Sinton asked whether the inclusion bodies were found in the lymphatic glands, as in cattle diagnosis.

Mr. Pay: Inclusion bodies certainly occurred in the lymph nodes, but he had never tried to identify them in smears from lymph tissue. Although they were generally to be found in histological sections of lymph node they were never as numerous or as easily recognised as those in the liver.

Col. Sinton asked if it would be possible to dissect out a lymphatic gland to make a diagnosis. This would be analogous to gland puncture in theileriasis to demonstrate Koch's blue bodies and might permit of an earlier and more accurate diagnosis.

Mr. Pay: He thought that in some cases a lymph node biopsy or even a liver biopsy would prove useful. However, it was important to bear in mind the extremely rapid course of the disease in fatal cases and the fact that a laboratory examination of such biopsy material would certainly take several days.

The President (Miss Joshua) said they would close the discussion at that stage. It had brought the problems of canine practice to the fore and probably, from that, advance would be rapid in the clinical field.

MOTION-STUDY TECHNIQUE APPLIED TO MEDICAL PRACTICE

In the current issue of the *British Medical Journal* there is an interesting account of how a doctor reorganised the clerical work of his practice with the help of the motion-study techniques often used in modern industry to simplify working methods and increase individual output.

A motion-study investigator observed and charted the activities of the doctor and secretary in planning a day's round and in keeping the normal records of visits. This showed that certain parts of the routine had become obsolete. By eliminating these, rearranging the work that remained, and designing equipment to simplify it, much redundant work was cut out and the new arrangements made it possible not only to get through the essential clerical work but also to do it more efficiently. The new system has also had certain good subsidiary effects. Records are now always up to date and from the cards on the rack it is possible to find the doctor when out on rounds.

The account in the *British Medical Journal* concludes: "It should be emphasised that this system is only the solution to one doctor's problem. It would need adaptation to suit a practice staffed in a different way. All motion-study improvements are made to suit individual circumstances and each problem must be studied without prejudice."

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The Fight Against Disease, which since 1921 has been the official organ of the Research Defence Society, has been renamed *Conquest*. Since the war only two issues per annum have been published; it is intended to publish *Conquest* quarterly, or even bi-monthly, and to include in it authoritative papers demonstrating important advances in prophylaxis, diagnosis and treatment, the relief of pain and the prolongation of life which have resulted from experiments on animals. The first number of *Conquest* includes a brief history of the society and a statement of its aims and objects.

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Weekly Wisdom

The fundamental principles of the new era are two in number, the veneration of knowledge and research and a regard for man's social well-being.—The Right Rev. E. W. Barnes, F.R.S., Evening Discourse at the 1950 meeting of the British Association.

VIRAL HEPATITIS OF DOGS (RUBARTH'S DISEASE)

I. Clinical and Pathological Observations on a Spontaneous Epidemic*

BY

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I. Introduction

The group of infectious fevers of non-bacterial origin which affect the dog, and commonly called distemper, consist almost certainly of a number of distinct aetiological entities. The original studies of Carré (1905), followed by the classical work of Dunkin & Laidlaw (1926), have drawn attention to at least one aetiological entity, that due to a virus which normally goes under the name of Laidlaw-Dunkin virus. More recently epidemics of a distemper-like disease (para-distemper) very similar to the disease due to Laidlaw-Dunkin virus, but characterised by a rather high incidence of neurological signs and of hyperkeratosis of the pads and nose, have been reported and referred to as "hard-pad" disease (Scheitlin, 1946; MacIntyre *et al.*, 1948); this probably represents an epidemiological entity, although the precise relation of the virus responsible to Laidlaw & Dunkin's virus is not yet established. More recently Rubarth (1947) in Sweden has published the results of an investigation lasting some 15 years which has drawn attention to the presence of yet a third aetiological entity in this group. This disease, called by Rubarth contagious hepatitis of dogs, would appear to be caused by a virus similar to that isolated in North America by Green and called by him fox encephalitis virus. Rubarth's original description of the disease, as well as the more recent ones of Riser (1948), Coffin (1948) and Chaddock & Carlson (1950) in the United States, are largely pathological and experimental; there seem to be few accounts of clinical and epidemiological observations of this disease, especially in a closed community.

The opportunity occurred to observe a spontaneous epidemic of hepatitis in isolated kennels, in which there was virtually no opportunity of dog-to-dog contact from outside and in which it was possible to allow the disease to run a natural course without treatment. Although sporadic cases have been recognised in Britain (Innes, 1949; Sheather, 1949; Montgomerie, 1950; Elmslie, 1950), few detailed observations have been published. The following is a preliminary account of some of the clinical, epidemiological and pathological observations which have been carried out on this epidemic.

II. Clinical Observations

1. History and Management

The community, part of the stud unit of the Canine Research Station, consisted of 38 dogs, comprising four greyhound bitches three years old and more, and 34 young dogs five to seven months old. The greyhound bitches had been in our possession three to 15 months, prior to which they had raced and had almost certainly been immunised against street distemper by the Laidlaw-Dunkin method. The 34 young dogs were three smooth-haired terriers, which had been on the premises since weaning and had recovered from an experimental infection with para-distemper virus obtained from another outbreak (Parry, 1950), four border collies here since weaning, and four greyhounds bred in the unit; the remaining 23 were red Irish setters, of which 19 had been bred on the premises and four had been received from a breeder when three months old. Careful record of any illness in these animals had been kept since they had been at the station. There had been an outbreak of leptospirosis (*L. icterohaemorrhagiae*) three months previously, when two deaths occurred and the remaining dogs were vaccinated with two doses of leptospiral vaccine (*L. icterohaemorrhagiae*) (Burroughs Wellcome). Otherwise, no illness had been observed. The young dogs were all being used for observations in connection with other investigations and had been subjected to routine eye examinations and night blindness tests. Several of the red Irish setters had shown abortive attacks of canine-hysteria during these tests.

The genetic constitution of these dogs was varied. The terriers and all the greyhounds were out-crosses; the collies were the product of a son-mother mating; of the setters, five were the product of a brother-sister mating and the remainder were products of three different out-crosses. *The nutriture and physique* of these dogs was excellent. They were well grown and without any significant physical defects. They were fed in troughs a mash, consisting of cooked whale meat and horseflesh, which was used to steep a finely ground cereal mixture, comprising oats 8 parts, wheat 2 parts, barley 1 part, and dredge corn, containing peas, beans and vetches 2 parts, to which small quantities of blood fibrinogen, whey concentrate, bone-meal and cod-liver oil were added. Some rusks were added to give a stiffer consistency. These ingredients were drawn from the same bulk supply that furnished the needs of the other dogs on the station. Each dog received daily approximately 190 grammes of the cereal mixture, 310 grammes of meat, 60 grammes of rusks and 25 grammes of whey concentrate, providing a calorie intake of 80 to 100 per kg. bodyweight per day. The animals had been in excellent health and making exceptionally good growth for some two months, during which the dietary regimen had been constant.

The stud unit was housed in premises a quarter of a mile from the nearest road, in warm huts with concrete floors and with earth runs outside, surrounded by a single 6-foot-high chain link fence, which had been occupied by dogs only within the previous six months. The young dogs were running together in one kennel with a communal lodging, while the four brood bitches occupied another. They were fed from a single kitchen by staff who handled dogs and utensils from both kennels; no attempt was made not to carry infection on clothes, hands or thermometers while making observations during the epidemic. During the epidemic a third kennel was brought into use to house the collies and terriers. These kennels were adjacent but not contiguous, and they were not surrounded by an outer dog- or vermin-proof fence and sanitary barrier. The staff handled no other animals on the station, but they did not change their clothes or take any special precautions when they arrived (special isolation measures had been planned, but the necessary facilities were not yet available).

There were other dogs at the station housed 200 yards away in a separate unit with its own staff. These dogs, 23 in number and mostly red Irish setters and greyhounds, were all over one year old and were mostly two years old and upward, excepting two sucking puppies. None of these animals showed any clinical evidence of ill-health.

* Received May 3rd, 1950.

2. THE CLINICAL SYNDROME

(a) *General Signs*

The first indication of any illness was a curious turpentine-like odour about the kennels, which the head kennelman, with 25 years' experience of foxhound kennels, reported as a "distemper smell," *i.e.*, one he had noticed frequently in outbreaks of "distemper." The following day several dogs were ill; several exhibited hysterical running-barking seizures and one had malaise, proceeding rapidly to prostration. However, perhaps the most prominent feature was the absence of evidence of ill-health on ordinary inspection and the very slight reduction of food intake. Dogs with a temperature as high as 106° showed only very slight malaise, so much so that the ordinary owner, or indeed a trained observer unfamiliar with these particular animals, would not have suspected illness in any but those very severely affected. The appetite remained good, although the food intake was reduced by about one-third. The coat retained its gloss; the animals were active and playful. There was no vomiting and no frank diarrhoea, although for several days the stools were soft, grey and pultaceous and slight straining occurred; there was no sign of blood in the stools, although blood did appear on the thermometer after rectal insertion in severe cases. This blood was a port wine colour without clots, mucus or desquamated mucous membrane. The animals remained alert with bright demeanour, except when circulatory collapse was imminent.

Ocular discharge was absent, except on the second morning only, when a purulent ocular discharge was present in five or six animals; it disappeared spontaneously within two or three hours and was not seen again. No signs of conjunctivitis were present, except in one case, which developed a transient kerato-conjunctivitis late in the course of the disease. Evidence of infection of the upper respiratory tract was conspicuous by its absence and a very slight purulent nasal discharge occurred in three cases only during convalescence and then for two to three days only.

On the fifth and sixth days nearly all the animals showed marked evidence of thirst and drank a great deal of water; this subsided within 48 hours, but reappeared during late convalescence, about the 15th to 16th day, for 24 to 36 hours. It was apparently unrelated to weather conditions and feeding.

The rectal temperature was raised and in the severer cases reached 104° to 106·5°. However, there were no clinical signs of ill-health during the first 24 hours of the hyperthermia, even when it was as high as 105·5°. *The cardiovascular system* exhibited small departures from normal, which were most apparent in the character of the pulse rather than in changes of its frequency. Tachycardia was not a constant sign, except in severe cases, while bradycardia occurred in three cases, in two of which it persisted for one to two days only. Arrhythmia occurred in three animals. A more accurate indication of the state of the disease was small changes in the character of the pulse; the pulse wave was reduced in amplitude, it became quick with an abrupt dicrotic descent and a wide pulse pressure. These changes were of a moderate degree only, except in severe cases, when they were quite obvious and were associated with collapsed peripheral veins, a very wide pulse pressure and a softness indicating a small wave amplitude during systole and small residual content in diastole. *The respiration* remained unaffected throughout and in no case was respiration other than a quiet movement of 16 to 20 frequency per minute. Palpation of the abdomen revealed no evidence of tenderness, except of mild degree in three very severe cases, all greyhounds, nor could any gross enlargement of the liver or spleen be detected. *The tonsils* were enlarged in six cases, several unilaterally, but this sign bore no relation to the severity of the illness. Signs of pharyngitis or laryngitis were absent.

Pallor of the mucous membranes was present in eleven cases and gave an indication of the severity of the disease, since the degree of pallor was parallel to the degree of disturbance of the pulse character. In severe cases the buccal mucous membrane became an ash-grey hue, while in two very severe cases, which died, icterus was also present. In one case two or three petechial haemorrhages occurred under the gums, while in three cases there was gross haemorrhage into the buccal cavity from the gingival margins of the upper molar teeth, usually the carnassial. In several cases severely affected, scale accumulated on the teeth very rapidly and disappeared on recovery.

Retraction of the eye-ball, with evidence of reduced intra-ocular tension on digital palpation, occurred in many animals on the fourth and fifth days of the epidemic. Coinciding with this, a number of animals showed some oedema of the conjunctival fold. Both these signs disappeared within 48 hours. The fundus and optic disc showed no abnormalities in seven animals examined carefully, even in those showing neurological signs. *Kerato-conjunctivitis* appeared suddenly in one case, after fever of 106° for five days; a diffuse general opacity of the substantia propria of the cornea with moderate injection of the conjunctival blood vessels developed in one eye overnight; 24 hours later the second eye was affected similarly, but the corneal opacities resolved without any vascular reaction, and in a further 24 hours both eyes were normal.

Hardening or hyperkeratosis of the pads or nose was absent, except in two cases in which there was moderate thickening of the base of the muzzle occurring about the eighth day, *i.e.*, during early convalescence. *Oedematous swelling* of the distal portion of one hind limb occurred in two cases during convalescence; in one case this persisted for 36 hours only, while in the other the oedema disappeared gradually over a week.

(b) *Neurological Signs.*

The hysterical seizures observed during the early stages of the epidemic were characteristic psychomotor seizures, as observed in canine-hysteria (Parry, 1949). The dogs run round wildly, often barking the while but always missing any solid obstacles, such as buckets, fence walls, etc. These dogs were nearly always attacked by the remaining animals. During the middle of the epidemic (four to six days) it was noticeable that a situation of moderate stress, such as a playful attack by three to four other dogs, which in health would have been playfully handled, became sufficient to initiate a frank outburst of hysterical psychomotor seizures, although prodromal anxiety was not present. A lessening of this group irascibility during play was one of the first signs of an abatement of the epidemic and heralded a general improvement. It was accompanied by the disappearance of the peculiar odour pervading the kennels.

Signs of disseminated encephalopathy occurred in one case. On the fifth day of the epidemic the dog was rather quiet and aloof, the pupils were constricted and the pupillary light reactions were absent; there was no fever and the dog was in other aspects clinically normal. On the next day the dog was markedly aggressive to its fellows if approached. Restraint for rectal temperature recording provoked a violent outburst of struggling with attempts to bite the handlers, which subsided immediately the animal

was released. A second attempt provoked a motor seizure; this commenced with champing movements of the jaw, gradually becoming more violent over a period of 15 seconds, when the head was lifted with the nose in the air, and the hind legs collapsed. In this posture jaw movements became very marked until the jaws were fully opened. The head was turned slowly towards the left shoulder, when the dog fell over on its right side. This initiated a stage of tonic spasm of the hind legs, head and neck, with fully opened jaws, lasting 15 to 20 seconds, during which the forelegs were flaccid; clonic uncoordinated foreleg movements followed for half a minute, during which the upper hind leg was flexed, but without any hypertonicity. Jaw movements continued throughout. At the end of two minutes the movements subsided and the dog was lying quietly; about half a minute later it rose, walked into the corner and forced its head to the ground with forelegs flexed and hind limbs extended. In this posture it remained quietly for some minutes, and permitted its temperature to be recorded (102·1°). Later it lay quietly in normal lateral recumbency.

Some eight to ten of these seizures were observed during the day, being precipitated by dogs barking or rough handling. They subsided when the animal was left in complete quietude. The dog took milk fairly readily, but not solid food.

On the third day of the neurological syndrome the dog was lying quietly, the pupils were normal in size and the light reactions had returned, but any handling of the animal provoked excitable movements. On the fourth day the animal was apparently quite normal, apart from very slight ataxia of the hind limbs and some sluggishness of the left patellar reflex. These signs disappeared on the fifth day and no evidence of the illness could be detected eight weeks later.

(c) Course of the Disease in Affected Animals

Of the 38 animals 22 showed some evidence of clinical ill-health, and of these 22, four died, five were affected severely, 12 affected mildly and two had aclinical infections, the significance of which remains in doubt. The criteria of severity, adopted on a purely arbitrary basis, were as follows. *Severe cases* showed slight but definite malaise on inspection; they lay about more than normal, and their playing was reduced, but rarely absent; there was evidence of bowel and/or gum haemorrhages; the cardiovascular system showed considerable changes in the character of the pulse which we associate with moderate to profound shock; hyperthermia from 104° to 106·5° was present; food intake was reduced, but never to less than one-half the normal; icterus and diffuse encephalopathy were inconstant signs. *Mild cases* showed such slight malaise that it would be recognised only by those well acquainted with the habits of the animals. There was very slight reduction of the food-intake and slight disturbances of the pulse characters, which could be detected with certainty only if normal animals of the same age and type were available for comparison. Hyperthermia might occur up to 103·5° to 104° for two to three days, but it was often of shorter duration. These dogs often showed hysterical seizures, which might be the principal sign. *Aclinical cases* were normal on inspection, food intake was unaffected and the only sign was hyperthermia of 1 to 2° F. up to 103° F., and persisting for 24 to 48 hours, with or without one or two hysterical seizures.

The four deaths all occurred in the first four days of the epidemic and in each case the duration of the clinical illness before death was 12 hours or less; one case in particular was perfectly normal at night and was found dead in the morning. The severe cases were ill for four to six days and then recovered rapidly and completely in two to three days. The mild cases were virtually normal within three to four days. None of the animals showed any marked loss of condition at the end of 14 days and no neurological sequelae have appeared after eight weeks.

3. Epidemiology

(a) Course of the Disease in the Community

The epidemic commenced during the first week in March, 1950, and continued for ten days. On the morning of the first day one greyhound showed some malaise and prostration with icterus, and one Irish setter had several hysterical seizures. During the afternoon the greyhound collapsed and died, while four more Irish setters were hysterical. On the second day six Irish setters were hysterical and were segregated, as the barking of the terriers, of which they usually took no notice, repeatedly precipitated mild running-barking seizures.

On the third day the dogs were all in apparently good health and no hysterical seizures were observed. On the morning of the fourth day a dog which was perfectly normal the night before was found dead. One dog showed severe illness in the late afternoon and died that evening. The faeces were grey and pultaceous, although on casual inspection very little abnormality could be detected in the dogs. On the fifth day many temperatures were slightly elevated and there was marked thirst. In general dogs were rather excitable and playing was apt to lead to fights. Some dogs lay about more than usual. The curious odour was particularly strong. On the sixth day the dogs were quieter and more content and playing was less likely to lead to fighting. Thirst was reduced and there was no hysteria. On the seventh day the odour was abating, but a number of dogs were observed to be straining for short periods, although the stools were rather firmer. Purulent nasal discharge appeared for the first time. On the eighth day the improvement continued and, apart from three dogs, there was no clinical illness. By the tenth day, food intake had almost returned to normal and a visitor would have been unaware that any of the dogs had been ill.

The progress of the epidemic is illustrated in the accompanying chart (Fig. 1—overleaf).

(b) Morbidity and Mortality

By the clinical criteria stated above, 22 of the 38 animals were affected (60 per cent.). However, since the disease was confined to the younger dogs less than 12 months old—none of the four aged brood bitches was affected—it seems more profitable to consider the morbidity and mortality rates in the younger group. Twenty-two of 34 animals were affected (67 per cent.), of which 12 were males and ten females. The incidence by breeds is 16 of 23 red Irish setters (70 per cent.), three of four greyhounds (75 per cent.), none of three terriers (0 per cent.), and three of four collies (75 per cent.). The mortality rate was four out of 34 (12 per cent.). These and other details are shown in Table I.

Several points are noteworthy. The terriers were apparently unaffected, as were the adult greyhounds. The morbidity rate in the other breeds was remarkably similar, but the number of severe cases was highest amongst the young greyhounds, who showed signs of more severe alimentary and cardiovascular disturbance than the other breeds. The mortality rate was also highest amongst the young greyhounds.

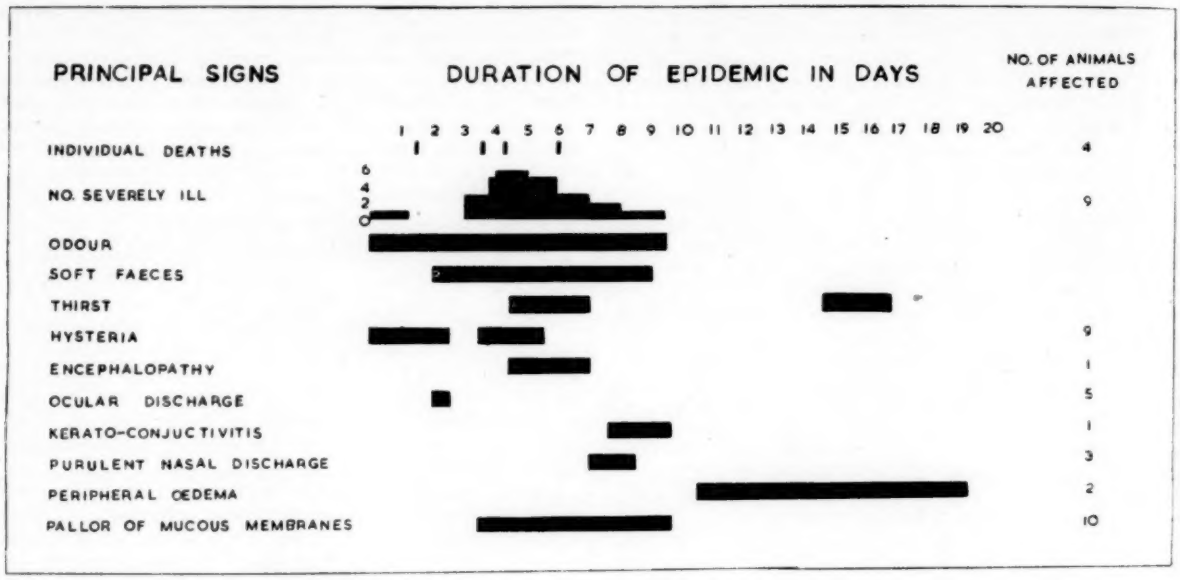

FIGURE 1

TABLE I

MORBIDITY AND MORTALITY RATES ACCORDING TO BREEDS

	Adult Grey-hounds	Young Grey-hounds	Red Irish Setters	Fox Terriers	Border Collies
Number originally present	4	4	23	3	4
Number affected	0	3 (75%)	16 (70%)	0	3 (75%)
Number severely affected	0	3 (75%)	5 (22%)	0	2 (50%)
Number of deaths ...	0	1 (25%)	3 (13%)	0	0

(c) *Incidence of Canine-Hysteria*

Hysterical seizures were observed in nine animals during the early phase of the epidemic and, indeed, were the chief presenting sign. The animals were all red Irish setters, three being females and six males. They were distributed fairly evenly between three litters, the progeny of the brother-sister mating having approximately the same morbidity as the two other litters; these latter had been observed to show evidence of mental instability during the night blindness tests. The one animal which had shown a hysterical seizure during the night blindness tests was not observed to be hysterical during the epidemic, but then it showed no clinical evidence of being affected.

III. Pathological Observations

1. Morbid Anatomy

Five animals came to autopsy, the four cases dying naturally and one being destroyed 48 hours after it had recovered from a mild attack of the disease. The *post-mortem* examinations were carried out within one hour of death in two cases and within 12 to 15 hours of death in the remaining three; the weather was cool (40° to 50° F.).

The mucous membranes exhibited marked pallor, the orifices were normal and *rigor mortis* might be present. On reflecting the skin there was abundance of subcutaneous fat, which was very white, all the skeletal muscles were pale, while in two cases there was also generalised icterus. All the carcase lymph nodes were enlarged and on section were markedly oedematous with congestion of the medulla. *The abdominal cavity* in three of the four fatal cases contained 300 to 500 ml. of a bloody fluid, which was apparently almost pure blood, being bright red in colour; over the omentum were enormous strands of red blood clot, extending from the ventral and diaphragmatic surface of the liver as far as the pelvis and lying particularly in the folds between the intestines. These strands of clot were 1 to 2 cm. wide and 0·5 cm. thick and in some cases near the liver their peripheral margins were of pure fibrin. On lifting the omentum, numerous areas of subperitoneal haemorrhages, 1 to 2 cm. in diameter, were apparent, adjacent to the main vessels in the mesentery, the wall of the intestines at their mesenteric attachment, in that portion of the mesentery surrounding the portal vein, and on the diaphragm adjacent to the liver. *The mesenteric lymph nodes* were enlarged to 8 cm. long by 1 cm. wide and deep; they were markedly oedematous with massive congestion confined chiefly to the medulla. The roots of the mesentery were distended by a clear serous fluid, while the mesenteric blood vessels and posterior vena cava were relatively empty of blood. *The liver* was enlarged and extended 2·5 cm. below the left costal margin; the diaphragmatic surface was covered with a thin film of fibrin which joined the individual lobes; the capsule was taut, although the free margins were not rounded; the surface had a regular mottled colour and granular texture, divided into zones about 1 mm. in diameter, each with a pale raised outer margin and depressed dark red centre, the whole organ being a brick red colour. On section, the substance of the liver was homogeneous, a light greyish-liver-brown, the blood vessels appeared relatively empty and the whole organ somewhat swollen. *The portal tract* was distended with a clear serous fluid which extended along the bile duct to the wall of *the gall-bladder*, which was grossly thickened, especially on the hepatic surface, where it was 1 cm. thick in parts. The bile was golden in colour and of a grumous consistency; the bile duct was patent. *The spleen* was swollen and tense, the margins rounded, and on section the pulp was a deep red " liver " colour, with the enlarged

malpighian corpuscles very prominent. The pancreas in two of the cases showed marked pallor.

The intestines were the site of severe damage. The contents of the intestines, which had the curious turpentine-like odour observed in the kennels, consisted throughout much of their length of almost pure blood, partly lysed and without clots. This was particularly apparent in the jejunum and ileum. The remainder of the contents were mucoid grey in appearance. The wall of the intestine was thickened and the mucous membrane oedematous. There were small zones of moderate congestion, but no evidence of serious damage to the mucosa. In general, the small intestine was contracted, but for several lengths 2 to 3 cm. long in the lower ileum, the lumen was dilated and here the congestion was more marked. The caecum was unaffected, although the patches of lymphoid tissue were prominent. The colon showed congestion of the wall, fresh blood in the lumen and portions of it were dilated; in the peracute cases the first (rostral) half was widely dilated, while the caudal half was constricted. *The wall of the stomach* was thickened and the mucous membrane was an ash-grey colour, but haemorrhages were uncommon (two in one case only). *The kidneys* were pale and on section showed little blood; in one case there was oedema of the pelvis. *The bladder* was almost empty and the urine amber coloured; there were no submucosal haemorrhages. The gonads and genital tract were normal. The pelvic lymph nodes were enlarged, oedematous, with congestion of the medulla as observed elsewhere. The pharynx was normal; *the tonsils* were slightly congested, and the oesophagus was normal.

The thoracic cavity had no excess fluid. There were subpleural petechial haemorrhages over the ribs 2 cm. dorsal to the costochondral junction in one case. The pericardial sac was normal. *The heart* was a curious parboiled colour, upon which the tracery of the coronary blood vessels stood out in marked relief. The cavities were small with a mixed " chicken-fat " and red clot on the right side, while the left side was almost empty. There was oedema at the base of the aortic valve cusps and one or two small subendothelial haemorrhages on the septum of the left ventricle. *The thoracic thymus* was grossly oedematous with distension of the interlobular connective tissue and with intralobular haemorrhages, while in two cases the substance of the gland was entirely haemorrhagic in the centre of each lobule. The anterior mediastinium was distended with serous fluid and the bronchial and mediastinal lymph nodes were enlarged, oedematous and with congested medullae. The larynx and trachea were normal, although in one case there was a suggestion that the vocal cords showed oedematous thickening. In two of the four cases the bronchi contained a little oedema fluid. The lungs showed some hypostatic congestion, but in one animal there was a haemorrhage in the cardiac lobe 2 cm. in diameter, surrounded by a suffused red zone.

The thyroid was pale and in one case two small petechial haemorrhages were present. *The adrenal glands* were very small and the medulla in particular was only 0·5 to 1·0 mm. wide with the cortex 1·5 mm.; they weighed 1·9 to 2·2 grammes each. *The pituitary* was not apparently abnormal, except that the posterior lobe appeared unusually friable and tore during removal of the brain, which it does not usually do so readily.

The meninges were pale, but otherwise normal. *The cerebro-spinal fluid* in the cisterna magna was rather excessive in amount and slightly turbid. The brain was rather pale and the gyri were nicely rounded. In all cases the brain filled the calvarium. *Cerebral vascular abnormalities* were present in one case only and were confined to the ventral aspect of the brain. In this case, small petechial haemorrhages were present on the pyriform lobes around the anterior cerebral artery, the ventral aspect of the left hippocampus and the extreme ventral lobe on the left side of the cerebellum; there was oedema around the vessels of the circle of Willis. The spinal cord was not examined beyond the second cervical vertebra. The brains were fixed whole and then cut transversely. *The ventricular system* was enlarged in three of the four brains examined. In two, one of which was from the dog killed in early convalescence, the posterior portion of the lateral ventricles was 1·5 cm. in diameter (the normal is 1·0 cm. or less) and the aqueduct of Sylvius 2.5 mm. diameter (the normal is about 1·5 mm.). In these three cases the small blood vessels of the brain stem were prominent and congested, but no macroscopic haemorrhages were apparent. The congested blood vessels were chiefly in the corpora striata, external capsules and the posterior portions of the caudate nuclei. The brain of one animal showed no macroscopic abnormalities. The optic nerves were normal, but on the right side in one animal there was a large haemorrhage around the optic nerve in the orbital fossa. The lymphoid nodules on the inner aspect of the third eyelid were conspicuous.

The bones and joints showed no gross abnormality. The femoral bone marrow examined in one case was bright red with a slightly greyer centre.

The four cases dying of natural causes showed a remarkable similarity, except in one respect. In the third case, the only one autopsied within one hour of death, there was no haemorrhagic effusion into the peritoneal cavity, but only some 50 ml. of straw-coloured fluid was present; there were no subserosal haemorrhages; the intestinal tract showed nothing on external inspection, but the wall of the small intestine was thick, and the lower colon only was congested with dark blood-stained contents.

The animal killed during early convalescence showed slight enlargement of the liver and spleen, slight oedematous swelling of some lymph nodes, no gall-bladder oedema and a normal adrenal gland (medulla 2·5 to 3 mm., cortex 1·5 to 2 mm. wide).

All five animals carried a moderate alimentary infestation with 30 to 40 young adult ascaris (*Toxascaris leonina*).

2. Histology

In impression smears made from scrapings of the liver surface and stained for inclusion bodies with Giemsa after methylalcohol fixation, the liver cells showed marked vacuolation of their cytoplasm, and the nuclei contained large basophilic inclusion bodies, which were round, homogeneous and up to half the nuclear diameter. There was pronounced margination of the nuclear chromatin. Paraffin sections showed the same changes in the hepatic nuclei, together with evidence of profound hepatitis.

IV. Microbiological Investigations

Specimens of liver, spleen, mesenteric lymph nodes, lungs, peritoneal fluid and heart blood were harvested at autopsy under aseptic conditions. No bacteria of significance were detected in these specimens on aerobic and anaerobic culture on Lemco, meat infusion, serum, liver and carbohydrate media.

Specimens of liver stored at –25° C. were ground up with saline to form a 10 per cent. suspension. The suspension was injected intraperitoneally into five dogs, four adults and one weanling; none of these animals showed any clear clinical

evidence of ill-health. Later, two ferrets were inoculated with another preparation of the liver, spleen and lung suspension, while two other ferrets (litter mates) and the weanling pup were inoculated with para-distemper virus (ferret spleen). The two former were unaffected, while the three latter all developed the usual signs of para-distemper.

No leptospira was observed in impression smears of liver stained with Giemsa or in sections of liver and kidney stained by Levaditi's method. Sera from three severely affected dogs and one unaffected dog, collected four weeks after the epidemic commenced, all gave a negative agglutination reaction against *L. icterohaemorrhagiae* and *L. canicola*.

Convalescent sera from the recovered dogs have shown an antigen-antibody reaction with liver antigen from fatal cases and also with liver antigen from a case of contagious canine hepatitis kindly supplied by Professor Rubarth of Stockholm.

V. An Interpretation of the Clinical and Pathological Findings

The disease process would appear to affect two organ systems preferentially, namely, the blood vessels and the liver. The signs of circulatory collapse with oligaemic shock, the excessive thirst, the oedema of the conjunctival fold and of the legs of the living animal, together with the very marked accumulation of fluid in many extravascular sites observed at *post-mortem* examination, all suggest that there is a profound disturbance of vascular permeability, particularly in the abdomen, and leading to passage of fluid from the blood stream into the extravascular spaces, followed in the severe cases by red blood cells. This would account in part for the pallor of mucous membranes seen in the severe cases and for the presence of blood in the intestinal lumen and the peritoneal cavity. The oedema surrounding the portal tract and extending to the wall of the gall-bladder is extremely striking, but in the case destroyed during early convalescence it was absent.

The liver damage is very extensive, although jaundice, which requires functional loss of some 90 per cent. of the total liver organ capacity before it appears, was observed in only two cases. However, histologically, the pathological changes are profound and widespread. It may well be that the shift of fluid to the extravascular spaces is accentuated by hepatic insufficiency with retention of anti-diuretic hormone and some degree of hypoproteinaemia.

The possible rôle of raised intracranial pressure in the pathogenesis of the neurological signs is of considerable interest. The slight excess of cerebro-spinal fluid and the dilation of the ventricular system suggest that a moderate communicating hydrocephalus was present with increased intracranial pressure. On the other hand, no papilloedema was detected even in severe cases with neurological signs. However, it is by no means certain that raised intracranial pressure does cause papilloedema in the dog (Wolff & Davies, 1931), as it does in many other species.

VI. Differential Diagnosis

The rapid death of the first case with acute oligaemic and haemorrhagic shock, icterus and petechial haemorrhages on the gums was suggestive of leptospirosis (Weil's disease), while the non-fatal cases in the greyhounds were reminiscent of so-called haemorrhagic gastro-enteritis of greyhounds. However, the autopsy findings were not typically those of leptospirosis or haemorrhagic gastro-enteritis, but were extremely similar to those of Hjarre's acute liver atrophy (Hjarre, 1930) and to severe cases of Rubarth's contagious hepatitis (Rubarth, 1947).

The syndrome differed in many significant respects from para-distemper. The fulminating onset and rapid recovery are quite different from that usually seen in para-distemper epidemics, while the clinical signs show many minor differences. The autopsy findings are different in that in para-distemper the liver is not enlarged, the spleen is contracted and empty of blood, evidence of excessive extravascular fluid is absent and the adrenal glands are enlarged, rather than reduced in size.

The significance of these differences was confirmed by the results of the animal inoculation. Young dogs and ferrets are highly susceptible to both the Laidlaw-Dunkin virus and to our strain of para-distemper virus, while material from this epidemic was not pathogenic for ferrets or for dogs, including one weanling pup, which was shown subsequently to be susceptible to our strain of para-distemper virus.

It is worth noting, perhaps, that one case dying very rapidly and autopsied within one hour of death showed lesions so inconspicuous that the disease would not have been suspected, unless cases with haemorrhagic effusions and other lesions had been seen from the same outbreak.

Diagnosis (Provisional). — Viral hepatitis, probably Rubarth's disease, based on the clinical findings, the autopsy lesions, the severe hepatitis with nuclear basophilic inclusion bodies, the absence of significant bacterial infection, and the animal infection spectrum. This diagnosis must remain tentative until the results of further infection and serological tests are to hand and a detailed examination of histological preparations has been completed.

VII. Discussion

The observations made in this outbreak set this syndrome apart from those of para-distemper observed in the last three years (Parry, 1950) and from the published accounts of the classical Laidlaw-Dunkin infections (Dunkin & Laidlaw, 1926). The fulminating onset, the very rapid rate of spread through the community with no new cases after about seven days, the absence of ocular and respiratory tract complications, the very acute development of the disease in some cases with death in less than 12 hours from the onset of ill-health, and the inconspicuous signs of the disease on casual inspection in the majority of animals, all serve to differentiate this syndrome. Whatever the final decision regarding the nature of the aetiology—and this must await more comprehensive investigations—the syndrome should always be considered as a possible diagnosis, when dealing with isolated cases of " distemper " or pyrexia of unknown origin (P.U.O.) in the dog. The very insignificance of the clinical signs in many of our cases suggests that this disease would go unrecognised if it occurred sporadically, unless opportunities occurred to carry out full pathological and microbiological investigations.

The occurrence of hysterical episodes as the presenting clinical sign in this outbreak is of interest, and bears out previous observations (Parry 1949, 1950) that hysterical episodes are not infrequent during the early febrile or prefebrile stage of virus infections in the dog. It is noteworthy that of the nine cases exhibiting hysteria only one was sufficiently severely affected to suggest ill-health on inspection and this animal died later; the remainder were mild cases and none has developed residual neurological defects. Occurring as sporadic cases these animals might well have been diagnosed as cases of idiopathic canine-hysteria, and attributed no doubt to dietary causes.

The case of acute disseminated encephalopathy might well be diagnosed as encephalitis. However, the rapid recovery of this animal without residual defects in the central nervous system suggests that the clinical signs were due to causes other than morphological damage. This interpretation is supported by the absence of histological lesions in the C.N.S. of some cases of para-distemper dying of a similar clinical syndrome (Parry, 1950). This case will be subjected to histopathological examination after a period to allow delayed neurological deficits to appear.

The sudden appearance of a diffuse non-vascular keratitis in one case and its very rapid disappearance is of interest in the light of a similar observation in an outbreak of para-distemper (Parry, 1950). It is not clear whether the corneal opacity is due to oedema of the stratum propria or whether it might be related to temporary trophic disturbances in the autonomic nervous system. In any event, it would appear to be quite unrelated to secondary infection.

The failure to set up clinical signs of infection in five dogs, and in two ferrets inoculated intraperitoneally with liver suspension, which may be due, of course, to the virus perishing in our material, is in line with the observations of both Green (1934) and Rubarth (1947); both these authors comment on the difficulty of setting up clinical infections in dogs regularly, even in weanling animals. This is in marked contrast to the great infectivity of para-distemper (" hard-pad ") virus for young dogs and ferrets, and the very high mortality rate in young dogs in natural epidemics (Parry, 1950). Both Green and Rubarth suggest that the susceptibility of dogs to the hepatitis virus is very variable and cannot be explained adequately on the basis of passive immunity acquired from the dam nor of an active immunity due to previous exposure. They both suggest that heritable constitutional factors are involved. Such an hypothesis would explain the wide variations noted in this epidemic from the severity of the illness amongst most of the greyhounds to the unaffected terriers and setters, and also our failure to set up clinical infection in dogs experimentally.

The predominant impressions of this epidemic are the inconspicuous nature of the illness even in severely affected animals, sudden deaths overnight without evidence of ill-health or at most for the previous few hours, and the very rapid course of the epidemic both in individual animals and through the community. Unless a bloody peritoneal effusion and oedema of the gall bladder, etc., are present, the autopsy findings are not striking and without laboratory aids may not be sufficient to determine the diagnosis. In fact, sporadic cases of the disease could pass unrecognised very readily.

VIII. Summary

1. An epidemic of an acute febrile illness in an isolated kennel of dogs is reported.

2. The clinical signs and autopsy findings indicate a profound disturbance of vascular permeability with severe hepatitis and lymph node involvement.

3. The presence of intranuclear inclusion bodies in hepatic cells, the infectivity spectrum in animals and the clinico-pathological findings all support a provisional diagnosis of vital hepatitis, probably Rubarth's disease.

4. The significance of the neurological signs and certain points of differentiation from para-distemper (" hard-pad " disease) are discussed

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FARM LAND LOSSES FROM BUILDING DEVELOPMENT

A forecast that agriculture may lose from 350,000 up to 2,250,000 acres in the next 20 years to development schemes was made by Dr. G. P. Wibberley at a Town and Country Planning summer school at Nottingham. And the loss between 1939 and 1970 would probably be 15 per cent. of the whole land area of England and Wales.

" What will be the effect on agriculture? The present expansion programme is achieving a 50 per cent. higher production over the 1938 level from about 750,000 acres less. If this increased level of output is to be maintained over the next 20 years it will have to come from an area of farmland further reduced by about another 750,000 acres."

The land likely to be taken consisted largely of the better farms with good permanent equipment, the replacement of which at present-day costs would be very expensive. This should be weighed in the balance along with the annual loss of output from the land itself. This emphasised the need for steering urban and other development on to the poorer grades of land and on to farms with poor capital equipment.—*Farmer and Stock-Breeder.*

* * * * *

IMPORTANCE OF POULTRY BREED TYPE

Various amendments to the Poultry Stock Improvement Plan Regulations for England and Wales have been suggested by the Poultry Association of Great Britain. It is felt that six-day week trapnesting with an average for the seventh day is a retrograde step and should not be provided for in the regulations. The Association maintains that the phrase " No particular standard of breed type need be observed " in the Regulations under breed type should be deleted. The Regulation for breeders' grades states that females used for breeding must have produced 170 eggs of at least 2 oz. during 48 consecutive weeks. The Association has suggested to the Ministry of Agriculture that this should be 160 for Sussex and dual-purpose breeds and 170 for all other breeds. Further, the period for all breeds should be 52 and not 48 consecutive weeks.

* * * * *

" HORSEMAN'S SUNDAY " SERVICE AT TATTENHAM

" Horseman's Sunday," inaugurated last year by the Horse and Pony Benefit Fund was celebrated again recently at Tattenham Corner. People arrived on horseback, in horse-drawn vehicles and in motor-cars, to attend a short service conducted by the Rev. A. Bird, vicar of Burgh Heath, at which a special blessing was sought for the horse and rider. After the service a display of *dressage* was given by Captain Skupinski, riding a five-year-old chestnut part-bred Arab mare by Jaleel. Finally the party walked in procession from the grandstand to the road, and each person received a rosette as a memento of the occasion.

In the last five years the Horse and Pony Benefit Fund has collected and distributed nearly £4,000.

CLINICAL COMMUNICATIONS

HEPATITIS CONTAGIOSA CANIS (RUBARTH)

JOAN O. JOSHUA, F.R.C.V.S.,
FINCHLEY, N.3

In view of the fact that diagnosis of cases of hepatitis contagiosa canis (Rubarth) in this country has so far been arrived at *post-mortem* when such examination has been called for in cases of unexpected death following a brief illness, or alternatively has been based on circumstantial evidence when more or less symptomless pyrexia has been observed in contacts with known hepatitis contagiosa canis cases, the clinical details of the two following cases, which occurred sporadically during the course of ordinary practice and in which it was possible to arrive at a fairly confident diagnosis during life, may be of interest.

CASE 1: *Subject.*—Five-year-old cocker spaniel, m.

History.—Illness of one week's duration. Symptoms at first merely diarrhoea. Appetite was maintained in the early stages but failed gradually until there was complete anorexia. There was progressive and increasingly profound dullness and some loss of weight. Excessive thirst was not noted. Vomiting occurred occasionally about the third to fifth day of illness but was not a particularly marked feature of the disease. Urine had been passed in the house on one or two occasions and had been noted by the owner to be deep orange in colour. Blood had been seen in the faeces.

Examination.—The dog was profoundly depressed and tended to stand about in a tucked-up posture. Normally a good-tempered dog, he showed definite resentment on handling, and particularly on being picked up. Temperature, 105° F. Mucous membranes were dirty and slightly pallid; one or two very small submucous haemorrhages were noted on the gums. Upward pressure in the region of the xiphi-sternum provoked a howl of pain. There was some general hyperaesthesia and the dog was obviously apprehensive of handling.

Diagnosis.—Hepatitis contagiosa canis.

Prognosis.—Grave.

Euthanasia was requested and carried out.

CASE 2. *Subject.*—Fifteen-month-old chow, m.

History.—Illness of four days' duration. Complete inappetence from the outset, very dull. Excessive thirst. No vomiting, diarrhoea or cough. Had shown resentment on being somewhat roughly handled the previous day. Defaecation in abeyance.

Examination.—The dog adopted a posture of profound depression with head and tail drooping and back slightly arched. He was hyperaesthetic to the point of being almost hysterical.

Temperature, 105° F. Impossible to assess mucous membranes owing to the pigmentation of the breed. There was evidence of recent and rapid loss of flesh.

Pressure over the area of the xiphi-sternum evoked screams of pain and provoked an hysterical outburst.

Diagnosis.—Hepatitis contagiosa canis.

Prognosis.—Grave.

Euthanasia was requested and carried out.

In both cases *post-mortem* examination showed typical gross pathology and detailed examination by Mr. T. W. F. Pay, of the Royal Veterinary College, confirmed the diagnosis.

REMARKS.—Both cases appear to have fitted in with a syndrome of acute hepatitis and not the sudden liver insufficiency as described by Craige; they were, on the other hand, rather more acute than the subacute hepatitis described by him.

Helpful diagnostic features in these cases were the profound depression, high temperature and the pain evoked by pressure in the xiphi-sternum area, this latter probably due to the changes in the gall bladder rather than the liver substance. The changed temperament was noteworthy, both dogs being known to be normally very good-tempered but showing an irritability amounting to a hyperaesthesia with consequent resentment on attempted handling.

REFERENCE

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* * * * *

A FURTHER CASE OF DEAFNESS FOLLOWING COAL GAS POISONING IN A DOG

NORMAN COMBEN, B.SC., M.R.C.V.S.

In 1949 I reported (*Vet. Rec.* **61.** 128) two cases of deafness in cats following poisoning with coal gas, and one case in a dog. Brief details of four other cases were presented. A further case has now come to my notice, as follows.

Subject.—Ten-year-old Collie bitch.

History.—The bitch had been left alone in a house from 1 p.m. until 11 p.m. on a warm day in June. When the owner returned there was a strong smell of gas in the room where the dog was sleeping, and gas was found to be escaping from an old meter about 7 ft. above the ground.

The bitch appeared to be unconscious, and although normally a very attentive and obedient animal did not stir when called. When she was touched, however, she roused herself and walked about slowly in a dazed manner. She showed no sign of hearing her owner or any noise, however loud.

Sequelae.—During 24 hours afterwards the bitch would not eat, was lethargic and wanted only to lie still. She appeared still to be dazed, but would drink water.

After 24 hours she made a complete recovery in all respects except as regards her sense of hearing.

After five days her sense of hearing appeared suddenly to return to normal, and it has remained normal since.

Acknowledgments.—I am indebted to Professor E. C. Amoroso for bringing this case to my notice, and to Mr. W. T. Price, the owner of the dog, also of the Royal Veterinary College, for providing the details of the case.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

SWINE FEVER:

Cheshire.—21, Richmond Grove, Cheadle Hulme, Stockport, Cheshire (Sept. 13th); Kenneth F. Hughes, Four Lane Ends Farm, Rainow, Macclesfield (Sept. 15th).

Derbyshire.—Bowshaw Farm, Dronfield, Derbyshire (Sept. 9th).

Hertfordshire.—Buttermilk Hall, near Buntingford, Herts (Sept. 12th).

Essex.—Alfred Buckenham, Steeple View Farm, Arterial Road, Laindon, Essex (Sept. 15th).

Kent.—College Meadows, Ospringe, Faversham, Kent (Sept. 14th).

Lincoln (Parts of Lindsey).—Bell Hole Farm, Kirton Lindsey, Gainsborough, Lincs; George Herbert Binks, Coneygarth, Haxey, Lincs; J. W. Couch, 36, High Street, Haxey, Lincs (Sept. 15th).

Northumberland.—Mickley Bank Farm, Stockfield, Northumberland (Sept. 11th).

Sussex-West.—Appleton House Farm, Drift Lane, Chidham, Chichester, Sussex (Sept. 14th).

Yorkshire, W.R.—Co-operative Slaughterhouse, Geldend Road, Leeds (premises of origin Stansfield Roal Piggeries, Kirkstall Road, Leeds); Moorhouse Farm, Allerton, Bradford, Yorkshire (Sept. 12th); 66, Edward Street, Sheffield, 3 (carcase at Sheffield Slaughterhouse Isolation Block) (Sept. 13th).

ABSTRACTS

[**Laparotomy in the Horse.** Donald, D. E. (1950.) *J. comp. Path.* **60.** 28–33. (1 table, 11 refs.)]

Twenty-seven intra-abdominal operations, 12 of them cryptorchid castrations, have been performed on horses *via* flank (21), midline (4) or paramedian (2) incisions. All but three healed by first intention. Chloral hydrate given intravenously as a 10 per cent. solution was the anaesthetic agent, but was not found to be entirely satisfactory for protracted operations.

Technique

Flank Laparotomy

Method A.—Skin, subcutaneous connective tissue, and aponeurosis of external oblique muscle are incised 6 to 7 in. in a downward and slightly forward direction. The internal oblique muscle is divided in the direction of its fibres, followed by the transverse abdominis fascia and peritoneum. The wound is closed in four layers: peritoneum and transversalis together, with No. O silk sutures placed ½ in. apart; internal oblique muscle with four to five interrupted linen sutures; fascia of external oblique and subcutaneous connective tissue also with linen; skin with interrupted No. 8 nylon sutures ¾ in. apart. Additional support is given by three vertical mattress sutures, placed equidistantly along the wound, which include the external oblique muscle. A gauze pad secured at its corners to the skin covers the wound area.

Method B.—The technique here is slightly modified. To increase the operative field it is necessary to section also the muscle portion of the external oblique. The internal oblique is incised across its fibres in the same direction as the skin incision, which necessitates ligation of a branch of the deep circumflex iliac artery. The external oblique muscle wound is closed by several figure of eight sutures.

Abdominal viscera which can be reached through flank incisions are:—

Right Flank.—The terminal duodenum, jejunum, ileum and ileocaecal junction, base of body and apex of caecum, the middle section of the right ventral colon, the middle portion of the small colon, the rectum, the bladder, the ovaries and uterus, the ductus deferens and internal inguinal ring, the bladder and umbilical folds, and the right kidney.

Left Flank.—The jejunum and anterior ileum, the small colon and rectum, the pelvic flexure and adjacent part of the left ventral colon (on two occasions the pelvic flexure has been approachable from right flank), the spleen, the left kidney, the middle portion of the small colon, the rectum, the bladder and umbilical folds, the ductus deferens and the internal inguinal ring.

Ventral Laparotomy

Method A.—A midline incision 7 to 8 in. long extending forward from slightly in front of the prepuce or mammary gland. The tunica flava is next incised in its midline and the linea alba is treated likewise. The peritoneum is incised and sutured to the edges of the linea alba using No. 0 silk and interrupted sutures 1 in. apart.

The linea alba incision with attached peritoneum is closed as one layer with interrupted linen sutures ½ in. apart. Four sutures of catgut No. 4 are now introduced at equal distances along the wound. "Each suture pierces the tunica flava ½ in. from its cut edge, passes through the mesial edge of the rectus muscle, then crosses over the peritoneal closure to enter the rectus of the opposite side and emerges ½ in. from the cut edge of the tunica flava." These four sutures are left untied until the linea alba-peritoneum layer has been oversewn with ½-in. ribbon gut, the sutures passing through the edges of the recti muscles and locked at every third stitch. Following the tying of the four gut sutures the tunica flava is closed with interrupted sutures of No. 2 catgut and the skin with No. 8 nylon. Finally, a sterile skin dressing is bandaged in position.

Method B.—A midline skin incision as in A exposes the tunica flava. The skin on one side is freed laterally for some distance from the underlying tunica flava and retracted. The tunica flava is incised over the middle of the rectus muscle and the muscle itself is divided in the direction of its fibres down to the inner rectus sheath, which is next incised along the same line of cleavage. The cut edges of the transversalis and peritoneum are kept together with forceps and are sutured as one layer with interrupted sutures of No. 0 silk ½ in. apart. The second layer consists of linen sutures ½ in. apart in the rectus muscle. The third layer is formed by a row of interrupted sutures of No. 2 catgut in the tunica flava. The skin is closed with interrupted sutures using No. 8 nylon, supported by three or four vertical mattress sutures of the same material to evert the wound edges.

Structures available.—The following structures are available for surgical purposes when the abdomen is entered by the ventral incision: the jejunum, the right and left ventral colons, the apex and part of body of caecum, the small colon, the rectum and bladder, the ovaries and uterus.

C. F.

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[**Shock: Its Production and Treatment.** Essex, H. E. (1950.) *Vet. Med.* **45.** 63–67. (18 refs.)]

In this paper the author, who has contributed extensively to our present knowledge of shock, discusses the subject from the experimental angle, under four headings.

Traumatic Shock was induced by manipulation of the intestinal tract by handling for 30 minutes, exposure and further handling every 15 to 30 minutes until shock and death occurred, the former indicated by a blood pressure equivalent to 70 m.m. of mercury. In the dog, a state of shock occurred most rapidly with ether anaesthesia, less rapidly with pentothal sodium and least rapidly with pentobarbital sodium. Apparently the enlarged spleen seen in animals anaesthetised with the barbiturates is due to storage of circulating erythrocytes which are gradually released with the elimination of the anaesthetic, giving an "auto-transfusion" and retarding the onset of shock. In the experiments conducted under ether anaesthesia atropine sulphate was found to be the most effective agent to delay the onset of shock.

The Toxaemia Theory of Shock gained little support from the experiments performed.

Haemorrhagic Shock.—Under ether anaesthesia 60 per cent. of the estimated blood volume in healthy dogs was withdrawn before death ensued, but it is nevertheless recognised that in sick animals a simple haemorrhage may be sufficient to precipitate a state of shock. The effect is due not so much to the actual quantity of blood lost as to the percentage of the circulating blood which is lost. Blood volume can be restored by blood transfusion or the administration of other fluids.

Shock resulting from the administration of Drugs and other Toxic Agents.—Symptoms resembling those of traumatic or anaphylactic shock can readily be produced in animals by substances such as epinephrine, histamine, snake venom, "D-L Substance" and extracts of Ascaris Serum.

It has been found with some of them that shock is due to spasm of the vascular bed of the liver or lungs and up to the present no adequate remedial measures have been evolved for this type of shock.

C. F.

NOTES AND NEWS

Diary of Events

Sept. 27th.—Meeting of the Editorial Committee, N.V.M.A., 1.30 p.m., to be followed by Meeting of the Executive.

Sept. 28th.—Summer Meeting of the East Midlands Division, N.V.M.A., at Thurgarton, Notts. (See Notice.)

Sept. 29th.—Meeting of the Midland Counties Division, N.V.M.A., at Cheltenham (Fleece Hotel), 2.30 p.m.

Oct. 3rd.—Meeting of the Crop Protection Panel of the Agriculture Group of the Society of Chemical Industry, at the Geological Society, Burlington House, Piccadilly, W.1. Dr. E. A. Parkin (Pest Infestation Laboratory, Slough): "Biological Tests of Insecticides for Stored Products Insects," 5 p.m. Visitors welcomed.

Oct. 5th.—Meeting of the Western Counties Division, N.V.M.A., at Exeter (Rougemont Hotel), 2 p.m.

Oct. 10th.—Scientific Meeting of the Zoological Society of London, Regent's Park, N.W.8, 5 p.m.

Oct. 13th.—General Meeting of the South Wales Division, N.V.M.A., at Carmarthen. (See Notice.)

Oct. 19th.—Meeting of the South-Eastern Division, N.V.M.A., at Tunbridge Wells (Royal Mount Ephraim Hotel), 2.30 p.m.

N.V.M.A. Council and Committee Meetings

The next Council and Committee Meetings of the Association will be held in London on October 30th, 31st, and November 1st. The time-table is as follows:—

*Monday, October 30th, at 36, Gordon Square, W.C.*1.
2 p.m.—Organising Committee.
4 p.m.—Home Appointments Committee.

*Tuesday, October 31st, at 36, Gordon Square, W.C.*1.
9.30 a.m.—Parliamentary and Public Relations Committee.
10.30 a.m.—Veterinary State Medicine Committee.
2.30 p.m.—General Purposes and Finance Committee.

*Wednesday, November 1st, at the Connaught Rooms, Great Queen Street (off Kingsway), W.C.*2.
11 a.m.—Council Meeting.

As announced below, an N.V.M.A. Conference on Artificial Insemination, open to members of the profession interested in this subject, will be held in the Auditorium of the Wellcome Research Institute on *Thursday, November 2nd*, at 11 a.m.

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N.V.M.A. Conference on Artificial Insemination

It is intended to hold a whole-day conference on artificial insemination, open to members of the veterinary profession interested in this subject. The conference will be held in the auditorium of the Wellcome Research Institute, 183, Euston Road, London, N.W.1, on Thursday, November 2nd, 1950, at 11 a.m. Members will remember that this was the venue of the very successful Grassland Conference in October, 1948.

The main topic of the conference will be consideration of the ways and means of effecting and maintaining an increase in the conception rate in herds receiving the artificial insemination service. Veterinary surgeons employed at A.I. centres and N.V.M.A. representatives on advisory committees will be invited. It is hoped that all other veterinary surgeons interested in artificial insemination will make it convenient to attend.

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PERSONAL

Appointment.—The appointment in the Colonial Veterinary Service is announced of Mr. R. S. Pinniger, M.R.C.V.S., as Veterinary Officer, Tanganyika.

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Births.—Mitchell.—On July 17th, 1950, at Bottoms House, Savile Road, Dewsbury, Yorks, to Freda (*née* Brown), wife of David Mitchell, M.R.C.V.S., a daughter—Pamela.

Stirling.—On September 5th, 1950, at Auckland, New Zealand, to Evelyn, wife of John Stirling, M.R.C.V.S., a daughter.

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Marriages.—Bowler—James.—On August 5th, 1950, at St. Mark's Church, Salisbury, Geoffrey Thomas Bowler, M.R.C.V.S., to Miss Vivienne Mary James, M.R.C.V.S. The following members of the profession were present: Messrs. J. B. White, E. Wallace, H. L. Pook, W. Foulston, G. S. Grater, A. M. Brown, F. G. Brown, E. J. Hughes and J. C. Whitney.

Stubbins—Murray.—On August 12th, 1950, at the Church of St. John the Baptist, Royston, Herts, the marriage took place between Alex G. J. Stubbins, M.R.C.V.S., and Margaret L. Murray, of Royston.

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R.C.V.S. OBITUARY

Crowhurst, Charles, 11, Peventon, Bodmin, Cornwall. Graduated London, December 14th, 1893. Died September 1st, 1950; aged 79 years.

Penhale, John Morley Liston, Senior Veterinary Research Officer, Ministry of Agriculture and Fisheries, of "Broad Oaks," Addlestone, Surrey. Graduated London, July 10th, 1913. Died at St. Peter's Hospital, Chertsey, September 9th, 1950; aged 62 years.

Robinson, Francis Edgar, Portway House Hotel, Frome, Somerset. Graduated Liverpool, December 10th, 1945. Died September 3rd, 1950; aged 27 years.

Mr. Charles Crowhurst, M.R.C.V.S.

Mr. Charles Crowhurst, whose death we record above with much regret, had practised in Bodmin for over 40 years. After qualifying at the London College, he followed his profession for some years in London and Birmingham, and later was in Government veterinary service in South Africa. He went to Bodmin in 1900, retiring in 1941, when Mr. A. V. H. G. Abbott succeeded him.

Mr. Crowhurst came of one of those families in which veterinary surgery is almost a traditional occupation. His father was the late Mr. James Crowhurst, F.R.C.V.S., of Canterbury, and his son, Mr. James Glanville Crowhurst, is in the service of the Animal Health Division of the Ministry of Agriculture. Other members of the profession were, or are, William James (brother), Charles and Usebius Gustavus (uncles), Frederick, Cecil, Gregory and John (cousins) and Arnold and Robert (nephews).

Outside his profession, in which he was well known throughout the Bodmin district, one of Mr. Charles Crowhurst's chief interests was in Bodmin Parish Church, at which he was a sidesman for many years and until recently a churchwarden. He was a keen horseman and rider to hounds with the North Cornwall Foxhounds and the Fowey Harriers.

His wife, who survives him, is a daughter of the late Mr. and Mrs. J. Julian Glanville, of Kirkland House. Deep sympathy is felt in her bereavement and that of the son, Mr. James Crowhurst, and daughter, whose husband, Mr. G. F. Widden, M.R.C.V.S., is in practice at Paddington.

The funeral service was conducted at the Parish Church by the Vicar (Canon G. W. S. Harmer), the family mourners including Mr. J. Crowhurst and Mrs. Widden, while Mr. A. V. H. G. Abbott was amongst the large assembly.

John Morley Liston Penhale

A Tribute

Those of us who were privileged to know John Penhale mourn his death just at a time when he had so well earned the due reward of a life's work in the service of the Ministry of Agriculture and Fisheries.

A native of Holsworthy, Devon, and one of the fifth generation of veterinary surgeons, Penhale joined the staff of the Diseases of Animals Branch in 1913, and served in various parts of the country before finally going to Weybridge in 1935 on promotion from Divisional Inspector for Devon.

In Devon, Penhale is remembered as a true and generous friend to all who sought his ready help and advice. His character was blended with his kindly nature and bore the mark of a gentleman. As a colleague he was loved and trusted. Sincere sympathy is felt with his widow and daughter—Barbara—in their loss. We salute him in passing.

R. R. W.

Mr. F. E. Robinson, M.R.C.V.S.

Much sadness will be caused his many friends in the profession by the death, at the early age of 27 years, of a very promising veterinarian in Mr. Francis E. Robinson, who died at the Bristol Royal Infirmary, where he had lain seriously ill for the previous 12 weeks.

The only son of Mr. and Mrs. F. Robinson, of Dovercourt, he went to Frome four and a half years ago to join the well-known Frome practice of Messrs. Stephens & Stephens.

On April 22nd of this year, he was married at St. John's Parish Church, Frome, to Miss Mavis Jones, the only daughter of Commander and Mrs. S. A. Jones, of Somerset Road, Frome. To her the deepest sympathy will be extended in her tragic bereavement. Mr. Robinson was taken ill only seven weeks after their marriage.

GENERAL OBITUARY

George Radbon

Another link with the old days at the London school has been broken, for "George" has passed on after a life time spent in the service of the College. I cannot discover with any precision at what date he entered its service, but he told me 'He reckoned that he was not much over 20 when he became assistant to 'Harry,' the slaughterman." As George was aged 78 when he died on August 28th, he must have worked for the institution for well over 50 years. George was a great character to whom nothing came amiss and whom no occasion could disturb. He met all his troubles with a laugh and, if necessary, fortified by a "quid" of tobacco. His was not the glory to ride the horses sent up for examination; his realm was in the back yard amongst the cattle, the goats and the sheep and in the old *post-mortem* room. His department was a veritable farmyard and he could tackle any job that came his way with great competence. But his duties were in no wise limited to the care of the occupants of his many boxes. He was responsible for all post-mortems, and sometimes there would be as many as five or six to be performed in a day on large animals without taking into account those carried out on the smaller and sometimes exotic beasts, and attention to the specimens sent up for toxicological report. He was responsible for the dissecting room, its fire and its heating system. He attended on Drs. Lauder and Clough in the Chemistry Department, did most of their cleaning and saw that there was a good fire in their private room. He did the rough chores in the Pathological Department and cared for the experimental animals. He was stoker for the large boiler which supplied the heating system of the Library, Pathological Practical Laboratory, Central Theatre, Museum and Biology Laboratory. He also gave a hand in the casting of large animals on the operating bed, and took his turn on night duty at the gate—complete with a very high collar and tie. Just, as it were, to fill in any odd time at his disposal he kept the large copper in the slaughterhouse going (the only supply of hot water in the College), he boiled out bones and assisted in the preparation of the specimens for dissection.

Now all these jobs, a task which would appal any modern young workman, he tackled with great efficiency. He never had any time to spend on idle contemplation or talk, but just kept hammering away, fortified with a quiet sense of humour and cheeriness which made light of any difficulty. To see him assist Sir John M'Fadyean at a *post-mortem* examination was a joy; and the repartee which passed between them on occasion was sparkling. Sir John would remark gently, "Oh, George, you should not have done that." George would reply, "Well, you told me to do it," or "Why didn't you say you did not want it cut." Quite often, too, he would break in to Sir John's dissertations if he thought that some salient feature had been missed or was not being given due regard. These two men, master and man, worked as a team and there were no repressions between them, for each recognised the worth of the other.

I have had many happy times with George. His stories of the College over the last 50 or 60 years could fill a book. His memories of the old professors, staff and students were such that I attempted to persuade him to jot down his reminiscences with a view to publication. He replied, "I could not do that, for I am no scholar." When I suggested that if he would only supply the rough notes I would set them in order and publish them under his name, he said "Oh, that would never do, for they would all say Old George never wrote that."

Just before the recent war George was taken ill. He underwent a serious abdominal operation, but it was found that his trouble was not a surgical one but pernicious anaemia. It looked as if he was doomed, but his indomitable spirit was such that he fought back and was able to resume light duty until 1942. He lived in his little cottage in College Grove through all the terrors of the blitz and was able to pay frequent visits to us until quite recently. After the war many of our men were new to the College and they were constantly being changed, so it happened that one Friday I was near the door when George came into the College to draw his pension. The porter, a new man, did not know George. He accosted him and asked what he was doing on the precincts of the College. George's reply was typical of the man, "You ask me what I am doing here? Why, I have been so long here that the College belongs to me and I belong to the College." What a great answer. It was such as one would expect from a good old faithful servant who had been accustomed to go about his master's business as if it were his own.

We took him to Highgate Cemetery on Friday, September 1st, and his old friends and colleagues, Stanley Bryant, "Bob" Burgess, Harry Pool and I attended to bid farewell on behalf of the College that he loved and served so well for so many years.

J. McC.

BOVINE ACTINOMYCOSIS

Mrs. D. Oxford is working in the Department of Bacteriology, Foresterhill, the University, Aberdeen, on the *Actinomyces* organisms causing actinomycosis in cattle.

Mrs. Oxford would like to receive material from such lesions in cattle and would be very grateful if practitioners would send specimens, particularly where pus containing the typical "sulphur granules" is present.

Specimens should be sent, in sterile, screw-capped jars, to Dr. J. W. Porteous, Department of Biological Chemistry, Marischal College, Aberdeen, from whom jars may be had on request.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

CYSTICERCUS BOVIS

Sir,—For some time past, in Blackpool we have been conducting a thorough investigation into the incidence of *Cysticercus bovis*, and a technique has been developed which includes multiple incisions into the inner and outer masseter muscles.

Our findings indicate that the average infestation of about 0·3 per cent. quoted in the second paragraph of the summary on page 479 of your issue of August 19th, 1950, is much below the actual incidences throughout the country, and for the benefit of your readers who are carrying out meat inspection, I think that the enclosed table would be of special interest.

Month		Bulls	Cows	Heifers	Steers	Total
1948	Slaughtered	5	470	63	111	649
December	Infected	Nil	8	1	Nil	9
	Per cent.	—	1·7	1·5	—	1·3
1949	Slaughtered	22	350	27	21	420
January	Infected	Nil	10	2	2	14
	Per cent.	—	2·8	7·5	9·5	3·3
February	Slaughtered	11	177	84	25	297
	Infected	2	7	3	4	16
	Per cent.	18·1	3·9	3·5	16	5·3
March	Slaughtered	13	171	52	74	310
	Infected	Nil	10	3	4	17
	Per cent.	—	5·8	5·7	5·4	5·4
April	Slaughtered	26	143	86	143	398
	Infected	1	10	2	17	30
	Per cent.	3·8	6·9	2·3	11·1	7·5
May	Slaughtered	28	140	149	316	633
	Infected	Nil	14	21	34	69
	Per cent.	—	10 ·	14·1	10·7	10·9
June	Slaughtered	28	191	33	106	358
	Infected	1	12	5	10	28
	Per cent.	3·5	6·2	15·1	9·4	7·8
July	Slaughtered	10	360	199	371	940
	Infected	Nil	30	17	26	73
	Per cent.	—	8·3	8·0	7	7·7
August	Slaughtered	10	563	236	524	1,333
	Infected	Nil	37	35	47	119
	Per cent.	—	6·5	14·8	8·9	8·1
September	Slaughtered	12	505	236	526	1,279
	Infected	Nil	32	53	23	108
	Per cent.	—	6·3	22·4	4·3	8·4
Total for	Slaughtered	165	3,070	1,165	2,217	6,617
ten months	Infected	4	170	142	166	483
Dec.-Sept.	Per cent.	2·4	5·5	12·1	7·4	7·29

In our view, the routine examination as now practised in many slaughtering establishments is quite insufficient, and there can be no doubt that if the Blackpool technique were practised throughout the whole of the country the figure of 0·3 per cent. mentioned by your contributors would be increased more than tenfold.

At the present time the percentage found by our meat inspectors at the abattoirs is still well over 6 per cent., and as the majority of the cysts are viable, it is in the interests of public health that a more detailed inspection, especially of the head, should be made by all engaged in this health service.

Considerable efforts have been made, with the co-operation of local authorities throughout Great Britain, to trace the sources of infestation, and from successful enquiries concerning 255 beasts, the following information was obtained:—

146 had been bred in Great Britain.

56 had been bred in Ireland with a stay of up to nine months in England and Scotland.

10 had been bred in Ireland with an indefinite stay in this country.

43 had been bred in Ireland and were forwarded direct to the slaughtering establishment.

These statistics indicate the wide area in the British Isles from which infested beasts originate, and the extent of the infestation is of such importance as to justify special research being undertaken by our veterinary colleagues into particular problems of reducing the incidence of the disease in animals.

Yours faithfully,
HARRY PRIESTLEY.

Municipal Health Centre,
Whitegate Drive,
Blackpool.

September 9th, 1950.

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F. HEPATICA AND THE TUBERCULIN REACTION

SIR,—I have recently seen references to the fact that cattle with *Fasciola hepatica* in the lungs will react positively to the intradermal and ophthalmic tuberculin tests. In these cases the lesions seen on *post-mortem* examination showed the presence of pyogenic organisms besides the flukes, but acid-fast bacteria were absent.

I should be interested to hear if any practitioners have come across similar cases.

Yours faithfully,
HENRY MERLEN.

Sapperton,
Cirencester, Glos.

September 7th, 1950.

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ATTESTATION: IS IT A GOOD POLICY?

SIR,—

SUMMARY

(1) By evidence supplied to *The Veterinary Record* on February 4th, 1950, and subsequent dates, human tuberculosis (pulmonary type) is increasing roughly in proportion to the decrease in bovine infection.

(2) The increase in human infection is due to an assumed deficiency in tuberculin content of milk which is obtained from a high proportion of tuberculin tested cattle which have given a negative result to the test.

(3) Tuberculin ingested frequently in milk produces a highly active passive immunity to human type bacillus, which immunity is kept active each time tuberculin subsidised milk is so ingested.

(4) Bovine tuberculin has a higher antibody value than human tuberculin because the bovine is the more virulent of the two types of bacilli.

(5) The artificial addition of tuberculin to milk is not practicable because of the impossibility of assessing amount of such tuberculin needed to elevate resistance to human type of organism.

The facts mentioned above are established.

(*a*) England. Falling tuberculosis incidence in human beings. Attested milk-producing cattle about 16 per cent.

(*b*) Scotland, Denmark, Jersey and Guernsey. Sharply rising tuberculosis incidence in human beings. Cattle tuberculin tested 70 to 100 per cent.

RECOMMENDATIONS

(1) Tuberculin testing of cattle should be illegalised by Act of Parliament.

(2) All cattle, milk producing and pre-parturient, should be clinically examined by a veterinary surgeon of the farmer's choice, each three months. This service should be superintended by the National Veterinary Medical Association. A County Veterinary Surgeon should be appointed by the N.V.M.A. for the purpose of forwarding instructions each month to practitioners in the county. It may be more economical for such an officer to superintend two counties, depending on the number of cattle existing in two adjacent counties.

(3) The veterinary surgeon would be responsible for all duties entrusted to him by the County Officer.

(4) All members of The Royal College of Veterinary Surgeons would be eligible for such an appointment one year after graduation.

(5) The cost of such a service would be recovered by N.V.M.A. from the Treasury.

My sincere thanks are due to you, Mr. Editor, for your patience and advice relating to these letters.

Yours faithfully,
L. PITCHER.

66, Coningsby Road,
High Wycombe.

August 12th, 1950.

[*This correspondence is now closed.—Editor.*]

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REPRESENTATION IN FORMATION OF N.V.M.A. POLICY

SIR,—From discussions at Cardiff it appears that some isolated members find considerable difficulty in playing their full part in the formation of policy within the N.V.M.A. Many assistants in rural areas are unable to attend meetings and are often unaware of issues which have been "referred to the Divisions" until it is too late to attempt to influence the decision. It is realised that there is no real substitute for personal attendance at Divisional meetings and taking part in the discussion, but I think it would help people whose circumstances make this impossible if the following procedures were adopted:—

Where possible a more detailed and precise agenda should be issued, giving particulars of any important subjects likely to be discussed under such headings as "correspondence." Provided this were received in good time it would enable members to send in their views in writing to the Secretary, or even to make special arrangements to attend when they felt particularly strongly on any subject.

Arrangements should be made for postal or proxy voting, at least for the election of officers and council, and possibly also for any important motions before the Division. These facilities are already available in some Divisions but not, I believe, in all.

A very brief account of the meeting might be circularised soon after it to those members who were unable to be present, provided they filled in and returned a tear-off request form attached to the notice convening the meeting.

I realise that all this would involve extra work for the long-suffering hon. secretaries, but if the numbers requiring the service were not large it would not be an impossible task, and if they were large it would be proof that such a service was really necessary, and special arrangements, such as the appointment of an assistant secretary, would be worth while.

Finally, I cannot help feeling that people often tend to grumble vaguely in private when by making an effort they could, even without attending meetings, get their grievances ventilated and their views considered if they wrote in about such things either to their divisional secretary or to headquarters.

On the few occasions when I have needed to consult the N.V.M.A. about a special problem, or make a suggestion, I have always found the officers extremely helpful and sympathetic, and I am quite sure they welcome enquiries and expressions of opinion from individual members.

Yours faithfully,
CONNIE M. FORD.

11, Domonic Drive,
New Eltham, London, S.E.9.

September 12th, 1950.